This is a digital copy of a book that was preserved for generations on library shelves before it was carefully scanned by Google as part of a project to make the world's books discoverable online.

It has survived long enough for the copyright to expire and the book to enter the public domain. A public domain book is one that was never subject to copyright or whose legal copyright term has expired. Whether a book is in the public domain may vary country to country. Public domain books are our gateways to the past, representing a wealth of history, culture and knowledge that's often difficult to discover.

Marks, notations and other marginalia present in the original volume will appear in this file - a reminder of this book's long journey from the publisher to a library and finally to you.

Usage guidelines

Google is proud to partner with libraries to digitize public domain materials and make them widely accessible. Public domain books belong to the public and we are merely their custodians. Nevertheless, this work is expensive, so in order to keep providing this resource, we have taken steps to prevent abuse by commercial parties, including placing technical restrictions on automated querying.

We also ask that you:

+ *Make non-commercial use of the files* We designed Google Book Search for use by individuals, and we request that you use these files for personal, non-commercial purposes.

+ *Refrain from automated querying* Do not send automated queries of any sort to Google's system: If you are conducting research on machine translation, optical character recognition or other areas where access to a large amount of text is helpful, please contact us. We encourage the use of public domain materials for these purposes and may be able to help.

+ *Maintain attribution* The Google "watermark" you see on each file is essential for informing people about this project and helping them find additional materials through Google Book Search. Please do not remove it.

+ *Keep it legal* Whatever your use, remember that you are responsible for ensuring that what you are doing is legal. Do not assume that just because we believe a book is in the public domain for users in the United States, that the work is also in the public domain for users in other countries. Whether a book is still in copyright varies from country to country, and we can't offer guidance on whether any specific use of any specific book is allowed. Please do not assume that a book's appearance in Google Book Search means it can be used in any manner anywhere in the world. Copyright infringement liability can be quite severe.

About Google Book Search

Google's mission is to organize the world's information and to make it universally accessible and useful. Google Book Search helps readers discover the world's books while helping authors and publishers reach new audiences. You can search through the full text of this book on the web at `http://books.google.com/`

CYCLOPÆDIA

OF THE

PRACTICE OF MEDICINE.

EDITED BY DR. H. VON ZIEMSSEN,
PROFESSOR OF CLINICAL MEDICINE IN MUNICH, BAVARIA.

VOL. II.

ACUTE INFECTIOUS DISEASES.

BY PROF. THOMAS of Leipzig, DR. CURSCHMANN of Berlin,
DR. ZUELZER of Berlin, PROF. HERTZ of Amsterdam,
AND PROF. VON ZIEMSSEN of Munich.

Translated by

JAMES C. WHITE, M.D., AND EDWARD WIGGLESWORTH, JR., M.D., of Boston;
EDWARD W. SCHAUFFLER, M.D., of Kansas City; AND A. BRAYTON BALL, M.D.,
J. HAVEN EMERSON, M.D., GEORGE H. FOX, M.D., EDWARD FRANKEL, M.D.,
AND JOHN C. JAY, JR., M.D., of New York.

ALBERT H. BUCK, M.D., NEW YORK,
EDITOR OF ENGLISH TRANSLATION.

BIBLIOTHECA · BODLEIANA

LONDON:
SAMPSON LOW, MARSTON, LOW, & SEARLE,
CROWN BUILDINGS, 188 FLEET STREET,
1875.

All rights reserved.

151. p. 72.

(*Entered at Stationers' Hall.*)

THE EDITOR takes pleasure in acknowledging the great assistance he has received from DR. FRANK P. FOSTER, in preparing the present volume for the press.

BIOGRAPHICAL SKETCHES OF THE AUTHORS.[1]

Louis Thomas was born January 22d, 1838, at Möckern, near Leipzig, where his father was a school-teacher. He was prepared for the "Gymnasium," by private tutors, and left that institution in 1855. He underwent his first medical examination in November, 1857, and in 1858 became Clerk of the Medical Clinic, which position he held until 1860. He was graduated in December, 1860, and was immediately appointed Assistant at the Surgical Clinic at Rostock, but in 1861 was made Assistant at the Medical Clinic at Leipzig. This position he held until 1865. During this period (1863 to 1865) he was Instructor in Medicine. In July, 1865, he was made Director of the Polyclinic at Leipzig, and in August, 1868, Professor. His writings on clinical subjects are numerous, and are contained in the "Archiv für Heilkunde."

Heinrich Curschmann, born June 28th, 1846, at Giessen, in Hessia, attended the Gymnasium of his native city, and studied at the University in 1863. He was graduated in 1868, after having served, during his last term, as Assistant at the Polyclinic. He was then appointed Assistant Physician of the City Hospital at Mayence, which included the position of Second Physician to the Invalid Hospital. During the great epidemic of small-pox in 1870 and 1871, which was very severe in Mayence, he was commissioned as city small-pox physician. After three years' service in these positions, he left Mayence, in order to prosecute his scientific studies in Berlin. Somewhat later he began practice in the latter city. His first scientific paper, histological in character, appeared in 1866, in Kölliker's "Zeitschrift für wissentsch. Zoologie." His later articles are partly of an experimental, and partly of a clinical character, and may be found in the "Archiv für klin. Medizin," and the "Archiv für Psychiatrie und Nervenkrankheiten."

Wilhelm Zuelzer, Instructor at the University of Berlin and Director of the Charité, was born at Breslau, November 10th, 1836. He studied in Breslau and Berlin, and, after passing his examinations, in Vienna and Paris.

---

[1] Very kindly furnished by Prof. von Ziemssen, for the American edition.

ERRATA.

VOL I.

On page 161, eighth line from top, for "trifacial" read "facial."

VOL. II.

On page 92, at the beginning of the fifth line from the bottom, a comma has been omitted after the word "bronchi."

On page 231, fifteenth line from the top, the word "chest" should be used instead of "heart."

On page 317, the word "modificaitons" should read "modifications.

On page 399. For formula for choral enema, read: Chloral, from one and a half to two drachms; gum arabic mucilage, water, each eight fluid ounces.

Also, for formula for Stokes's Cognac mixture, read: Best brandy, distilled water, each two fluid ounces; the yolk of one egg; syrup, one fluid ounce.

On page 538, sixth line from the top, for "(1:50–60)" read "(one grain to the fluid drachm.)"

On page 662, foot-note, for "pills............doses," read "pills containing equal parts of sulphate of cinchona and saccharated carbonate of iron, of which from twenty-three to thirty grains are taken through the day in hourly doses."

On page 663, sixth line from the top, for "180" read "185"; seventh line from the bottom, for "Barnatzik" read "Bernatzik."

On page 664, last line of foot-note, for "(10 lbs. 8 ounces to the bath)" read "(one hundred and twenty-nine American, or one hundred and forty-one British ounces to the bath)."

On page 665, second line from the bottom, for "chinoidine" read "quinoidine;" in second foot-note, for "Quinodine" read "Quinoidine."

On page 670, eleventh line from the top, for "180" read "185."

On page 677, in the foot-note, for "bebeerine" read "berberine"; and for "3½ to 15 grains" read "from three to fifteen grains."

On page 737, at the top, for "narcottic" read "narcotic."

CONTENTS.

THOMAS.

VARICELLA, MEASLES, RUBEOLA, AND SCARLET FEVER.

PAGE

Introduction 3

VARICELLA 5

History 6

Etiology 8

Pathology 10

Anatomical changes 10

Symptomatology 15

Diagnosis 22

Prognosis 29

Complications 29

Treatment 30

MEASLES 31

History 36

Etiology 37

Pathology 62

Anatomical changes 62

Complications 90

Sequelæ 102

Diagnosis 106

Prognosis 109

Treatment 116

RUBEOLA.

(Rötheln, German Measles.)

Historical notice 130

Etiology 134

Symptomatology 141

Complications 147

Prognosis 147

Treatment 147

(Translated by Edward Wigglesworth, Jr., M.D.)

SCARLET FEVER.

Introductory remarks 158

History 159

Etiology 161

PAGE
- *Pathology* 202
  - Anatomical changes 202
  - Different forms of scarlet fever 234
  - Course of the disease in its usual forms 237
  - Affections of the lymphatic glands 242
  - Affections of the joints 243
  - Scarlatinal nephritis 244
  - Irregular course of scarlet fever 250
- *Complications* 274
- *Sequelæ* 283
- *Diagnosis* 284

(Translated by Edward Frankel, M.D.)

- *Prognosis* 287
- *Treatment* 297

(Translated by John C. Jay, Jr., M.D.)

CURSCHMANN.

SMALL-POX.

(Variola, Varioloid, and other modifications.)

- *History* 320
- *Etiology* 325
- *Pathology* 340
- *Symptoms and course of the disease* 340
  - Regular course of variola vera 355
  - Variola confluens 366
  - Variola hemorrhagica pustulosa 369
  - Varioloid 372
- *Complications and sequelæ* 378
- *Anatomy* 380
- *Diagnosis* 388
- *Prognosis* 391
- *Treatment and prophylaxis* 395
  - Vaccination 401

(Translated by George H. Fox, M.D.)

ZUELZER.

ERYSIPELAS, MILIARY FEVER, DENGUE, INFLUENZA, AND HAY FEVER.

ERYSIPELAS.

- *Definition* 423
- *History* 423
- *Etiology* 425
  - Exciting causes 432
- *Pathology* 441
  - Anatomical changes 443

PAGE
Symptomatology 451
Complications and sequelæ 465
Diagnosis 470
Stages and duration 472
Relapses 473
Varieties 474
Mortality and prognosis 476
Treatment 477

MILIARY FEVER.
Introductory remarks 486
History and epidemiology 487
Etiology 491
Anatomical changes 495
Symptomatology 497
Diagnosis 500
Mortality and prognosis 501
Treatment 502

(Translated by James C. White, M.D.)

DENGUE.
Definition 506
History and etiology 507
Symptomatology 510
Treatment 514

INFLUENZA.
Definition 516
History 517
Etiology 522
Pathology 527
Symptomatology 528
Diagnosis 535
Mortality and prognosis 536
Treatment 536

HAY FEVER.
Introductory remarks 540
Etiology 542
Symptomatology 547
Diagnosis 551
Treatment 551

(Translated by J. Haven Emerson, M.D.)

HERTZ.

MALARIAL DISEASES.
History 557
Geographical distribution 559
Etiology 563

PAGE
Pathology 587
General course of the disease 588
  Simple intermittent 588
  Masked fevers 598
  Pernicious fevers 602
  Remittent and continued malarial fevers 614
  Chronic malarial infection 620
Pathological anatomy 624
Analysis of individual symptoms 630
Course and sequelæ of the disease 647
Prognosis 650
Diagnosis 652
Treatment 655

(Translated by Edward W. Schauffler, M.D.)

VON ZIEMSSEN.

EPIDEMIC CEREBRO-SPINAL MENINGITIS.

Introduction 687
History 687
Etiology 690
Pathology 697
  General description of the disease 697
Pathological anatomy 703
Analysis of individual symptoms 707
Complications and sequelæ 721
Diagnosis 730
Course, duration, and terminations 732
Prognosis 733
Treatment 733

(Translated by A. Brayton Ball, M.D

VARICELLA, MEASLES, RUBEOLA,

AND

SCARLET FEVER.

THOMAS.

INTRODUCTION.

THE conception of acute exanthematic diseases, in the sense of specific morbid processes, is an abstraction belonging to recent times; the physicians of antiquity and of the middle ages, so far as they paid any attention to the occurrence of acute exanthems, regarded these as merely individual manifestations of that "pestilent fever" under which they classified no small number of epidemic diseases,—a way of looking at morbid phenomena which renders it difficult, and often impossible, for the investigator of modern times to profit by the records of those observers, and to derive any exact information with reference to the manifestations of the acute exanthems, as they occurred at that time.

The term acute exanthems is applied to those infectious diseases characterized by a regular sequence of individual manifestations which arise from a specific contagion, and are distinguished by typical morbid appearances of the skin. These properly comprise Measles, Rubeola, Varicella, Scarlet Fever, Variola, and Vaccinia. As to Erysipelas, doubts may be still entertained.

The common character, especially of the first-named diseases, expresses itself in the facts, I. That a feverish condition precedes or accompanies the appearances upon the skin, with the exception at most of the lightest cases; II. That the appearances upon the skin are developed in a definite order, at definite periods; III. That the participation of the organism, as a whole, is shown by the occurrence of definite morbid phenomena proceeding from various organs and systems; IV. That these diseases, with rare exceptions, attack the same person but once.

For the sake of clearness, it is customary to divide the course of the acute exanthems into single stages, and to distinguish: I.

The stage of incubation, of latency, or of the latent affection (these two terms not being always properly applicable), from the moment of infection to the beginning of well-marked and generally feverish symptoms; II. The prodromal or precursory stage, stad. prodromorum, from the commencement of positive disease to the distinct appearance of the typical eruption; III. The stage of eruption, that of the forming and formed exanthem, divided accordingly by many, for the sake of distinction, into the stages of eruption and florescence; IV. The stage of retrocession of the essential exanthematic process (stad. desquamationis, exsiccationis, decrustationis), which begins as soon as the exanthem has passed its maximum of development, and convalescence occurs, recognizable by the disappearance of all local and general (febrile) symptoms.

VARICELLA.

Vidus Vidius, Ars univ. med. Venet., 1596. P. II. L. XIII. cap. 6.—*Sennert*, Med. pract. l. IV. c. 12.—*Rivière*, Prax. med. Lugd., 1660. T. II. p. 685.—*Diemerbroek*, De variolis et morbillis, c. 2.—*Heberden*, Med. Transact. of the Coll. of Phys. Vol. I., 1767, p. 427. Comment de morb., c. 96.—*Willan*, Ueb. d. Kuhpockenimpf., übers. v. Muhry, p. 62.—*Heim*, Horn's Arch. 1809. Bd. X. pag. 183. Ib., 1825, pag. 1.—*Hufeland*, Huf. Journ. 1824. 4. Oct. p. 19. —*Thomson*, An Account of the Varioloid Epidemic, etc. London, 1820.—*Hesse*, Ueb. Varicellen, etc. Leipzig, 1829.—*Schönlein's* Path. 5. Aufl. 1841. II. p. 247.—*Canstatt*, Handb. d. med. Klin. II. p. 76. Erlangen, 1847.—*Löwenhardt*, Cstt. Jahresb. 1850. IV. p. 144.—*Trousseau*, Med. Klin. übers. von *Culmann*. I. p. 89. Gaz. des Hôp. 1842, p. 147. Union méd. 1850, p. 479.—*Delpech*, Journ. de Méd. 1846, p. 1, 33.—*Simon*, Hautkr. Berl., 1848.—*Bärensprung*, Arch. d. V. f. g. Arb. II. p. 308, 1856.—*Gintrac*, Journ. de Méd. de Bord. im Journ. f. Kinderkrankh. 1858, Bd. XXX. p. 221.—*Hebra* in Virch. sp. Path. Bd. III. 1. Aufl. 1860. 2. Aufl. 1872.—*Küttlinger*, Bay. Intelligenzbl. VII. p. 35, 1860.—*Tüngel*, Klin. Mitth. f. 1858. Hamb., 1860, p. 27.—*Vetter*, Arch. d. Heilk. 1860. I. p. 286. Virch. Arch. Bd. XXXI. p. 401.—*Plagge*, Memorab. IX. p. 78.—*Hauner*, Beitr. z. Pädiatr. 1863, p. 54.—*Thomas*, Arch. d. Heilk. VIII. p. 376. Memorab. XV. p. 209. Arch. f. Derm., I. p. 329.—*Locher*, Krankh. d. Haut. Erl., 1867, p. 161.—*Bockshammer*, Würt. Corr. 1867, p. 243.—*Ranke*, Arch. f. Derm. I. p. 99.—*Güntz*, Ib., p. 633.—*Körber*, Pet. med. Zeitschr. XIII. p. 319—*Förster*, Jahr. f. Kindhlk. N. F. I. p. 131. —*Henoch*, Virchow-H. Jahresb. f. 1868. II. p. 642.—*Czakert*, Zeitschr. d. Wundärzte Oesterr. 1869. No. 49.—*L. Meyer*, D. Klin. 1870. Nr. 6 ff.—*Eisenschitz*, Jahrb. f. Kindhk. N. F. IV. p. 205.—*Fleischmann*, Ib. III. p. 444. Wien. med. Woch. 1870. Nr. 51. Arch. f. Derm. III. p. 497.—*Kaposi*, Arch. f. Derm. 1873. V. p. 255.—*Kassowitz*, Jahrb. f. Kinderhk. VI. p. 160.—*Douillard*, Un. méd. 139, 1872. Jbch. f. Khk. VI. p. 187. 1873. *West*, Kindkh. 5. Aufl. Deutsch von *Henoch*, 1872.—*Obermeier*, V.-H. Jber. 1872. II. p. 272.—*Cantani*, Ibid. p. 272.—*Boeck*, Norsk Mag. for Laegevid. 3. R. III. 10. Ges. Verh. p. 126, 1873.—*Steiner*, Kinderkh. 2. Aufl. 1873.—*Küster*, Kunze's Zeitschr. f. pr. Med. 1874, p. 11.

THERE can be no doubt of the propriety of regarding Varicella as a peculiar and independent form of disease. It is true that this is still contested by physicians of eminence, but upon such unstable and easily controvertible grounds that it seems almost wonderful that these can be so repeatedly brought forward. It is pre-eminently variola with which varicella is confounded, and it is to the lightest forms of that disease that the name varicella is applied by the opponents of a specific varicella. It is therefore necessary, in reading about varicella, to always understand clearly whether the author believes or doubts the independent nature of varicella; since heedlessness in this respect has already given rise to very serious confusion.

HISTORY.

Varicella appeared first, according to Hesse's elaborate treatise (1829), under the title of Crystalli, and received by degrees a multitude of different appellations, such as variolæ nothæ, spuriæ, illegitimæ, hybridæ, crystallinæ, pseudovariolæ, false variola, pointed-pox, wind-pox, water-pox, chicken-pox, etc., that is, appellations pointing universally to a certain resemblance to variola. The most usual and appropriate name is varicella, and this is now universally employed by the upholders of its specific nature, coupled at most with the adjective "true" to distinguish it from the light form of variola, for which—to avoid confusion—the use of some other name, on the part of the adherents to the identity of the two, would be most desirable.

Varicella appears to have been known in old times, but to have been but little regarded, on account of its mildness; still, undoubted evidence of this does not exist. The first one in Germany to give an exact description of it was Sennert; in France, Rivière (1660) is regarded as the first one who gave it a careful investigation; in England this pre-eminence belongs to Harvey (1696), and in Holland to Diemerbroek. In general, however, but little was known of varicella until the time when the inoculation of variola began to prevail. When, for instance, the opponents of this reproached it continually with not affording to those inoculated a sure protection against the infection of natural variola, they frequently committed the error of regarding as true variola an attack of varicella, occurring after the inoculation; and *vice versâ*, the defenders of inoculation regarded as varicella light subsequent attacks of variola. It became, therefore, quite necessary to establish a more accurate differential diagnosis between variola and varicella, and especially to pay more heed to the former. In every respect that view is entirely erroneous which regards varicella as having first appeared since the inoculation of variola,—a view which is based upon the fact of the continual confounding of varicella with light forms of variola. Heberden (1767) was the first to insist upon a more exact distinction between the two diseases, and his work excited, of course, great opposition. Universal attention, however, was only then directed to varicella, after the introduction of vaccination, when repeatedly cases of variola were observed recurring in persons who had been vaccinated. Here also it was often the case that an attack of true varicella was regarded as variola, and especially that a variola occurring after vaccination showed, in many points, a closer resemblance to varicella than the variola of an unvaccinated person. A more careful study of varicella was now undertaken by Willan, and in particular by Heim. This decided advocate of its specific character pronounced at first all variolous affections of vaccinated persons to be varicella, or if such an one were really variola, that it was due to imperfect vaccination, and he sought, therefore, in particular, to establish a means of differential diagnosis between the two; even later, when compelled to admit that a person might have variola, after proper vaccination, he in no way changed his opinion in regard to the specific nature of varicella. It is due to Heim's influence that the individuality of varicella has been maintained in Germany; while in England Thomson's active advocacy of the identity of the contagious element in variola and varicella, added to the increasing lack of confidence in the inoculation of cow-pox virus, on account of frequent attacks occurring in vaccinated persons, has produced a certain perplexity and indifference in regard to the diagnosis of varicella, and consequently a neglect of it. The case was similar in France. A little later, indecision began to appear also in Germany. Hebra, namely, declared roundly, in spite of all opposing facts, that varicella was the lightest form of variola, and maintains this convenient though incorrect position with inconceivable persistency to the present day; no wonder, considering the authority which the teacher of dermatology at the Vienna University with good right possesses, that his opinion has found new adherents. The grounds adduced by him and by his scholars will be discussed when we come to consider the symptoms. Notwithstanding this, it may be assumed that the majority of physicians in Germany who are specially interested in children's diseases, or who are clinical teachers, uphold the separate nature of varicella, a belief which has been especially supported by Trousseau, in France, but has not, as it seems, met with universal acceptance in England.

ETIOLOGY.

Varicella is a disease of childhood, and attacks by preference young children, and even sucklings. In children over ten years of age attacks are infrequent, and I never saw an adult suffering from varicella. Yet it cannot, of course, be maintained with certainty that every predisposition to it is absent in the case of older children and of adults, for so wide-spread is the disease, that these may possibly have had it in their earliest childhood and no impression have been left upon either their memories or their skins, and in this manner the individual predisposition have been destroyed. But at all events it is very striking that, in contrast with the measles, which by no means unfrequently attacks even adults, varicella is rare even in the case of children of advanced years, and it may be that the correct explanation is that of diminished susceptibility after the tenth year, and its cessation at the time of puberty. In favor of this is the fact that the few maladies of elderly children are, as a rule, of but moderate severity. The explanation given by Kassowitz—a believer in the identity of variola and varicella—of the circumstance that young children are almost never attacked by varicella, is excessively forced. He attributes it to the greater delicacy of the young skin, and especially to the thinness of the epidermis, which permits the local inflammatory process—in varicella a less intense one at all events than in variola—to express itself by the extremely rapid formation of a serous fluid, the evaporation of which allows a much more rapid disappearance of the local trouble. This is readily disproved, inasmuch as an adult with a thick skin has never contracted a variola from a properly diagnosticated case of varicella in a child,—an occurrence which must needs have taken place were the two identical. Furthermore, it is well known that unborn children—even where the mother escapes the disease—readily fall ill of true variola, in spite of their very delicate skins, while no child has ever been born with true varicella. This is probably due in part to the absence of susceptibility to varicella in the mother, the necessary vehicle of infection.

Varicella occurs, according to the indisputable testimony of country physicians, at one time sporadically, at another as a moderate epidemic, without any connection with cases of variola. In large cities such epidemics occur, not after the manner however of epidemics of variola or measles, namely, at tolerably regular intervals of several years, but almost annually, or even twice a year,—with us, for example, with great regularity shortly after the opening of the "Kindergärten." Their extent and intensity are consequently never remarkable, nor their increase and retrocession and general course so characteristic as in the other two diseases. While variola often diminishes and seems almost to disappear for years at a time (at least after systematic vaccination), individual cases of varicella are always present, though perhaps not brought to the notice of physicians from the slight inconvenience they occasion. The needful conditions for the dissemination of varicella exist without doubt throughout the whole year, which cannot be the case with variola, since this is observed especially in the spring months. Its contagion is, like that of scarlet fever, endemic in thickly populated districts.

With regard to the vehicle of the contagion nothing is known absolutely; it is supposed that the communication takes place chiefly through the organs of respiration. Its infecting power, however, does not appear to be great, which explains, in part at least, the limited spread of its epidemics.

The contents of the varicella vesicles must, in contrast with those of variola pustules, be regarded as, practically, not inoculable. According to Hesse's compilation, inoculation produced no result in eighty-seven cases, in seventeen was followed by a merely local, and in nine by a general eruption. Apart from the fact that in most of the cases a negative result was obtained, it may be remarked with regard to the successful inoculations, that almost all of them possess these elements of doubt, viz., they do not exclude the possibility of spontaneous transmission in the usual way; in the next place the nature of the resulting eruption suggests the probability that the varicella virus takes no part in its genesis; and, finally, it should be remembered that where the results were merely local, no further attempt was made at inoculation, as especially noticed by Wunderlich. The contents of well-marked varicella vesicles have been inoculated with negative results by Heim, Vetter, Czakert, Fleischmann, and myself. This of course does not controvert the experience which proves varicella to be contagious, but merely the possibility of transmission by the method employed. Perhaps the failure was due to the small amount of matter inserted; perhaps the contagious properties are not in an active condition in the clear contents of the vesicles, which of late have been exclusively employed to prevent interchanges with the matter of variola; perhaps the incision exercises an injurious influence upon the inoculation;—these are points, concerning the nature of which we know as little as we do of the contagion itself. It is also possible that in many cases the peculiar susceptibility necessary to the formation of disease products has been absent, thus preventing the possibility of a varicella eruption. In marked contrast with the difficulty, or even impossibility of an inoculation of varicella, stands the facility of its dissemination among little children and its capability of infection.

PATHOLOGY.

Anatomical Changes.

The exanthem of varicella is, in its appearance as a whole, so characteristic that the disease can hardly be mistaken if seen at its acme of development. A false diagnosis only results, as a rule, from the fact that the patient is seen for the first time when the eruption is rapidly disappearing. The eruption consists of a varying number of vesicles, from the size of a pin's head to that of a large pea, or even greater, mostly round, and seated, in marked contrast with the pustules of variola, very superficially. They become very exceptionally, and only in isolated spots, true bullæ, from the size of a dime to that of a silver dollar, but, as a rule, retain nearly their original dimensions. They vary in number as a rule from two hundred to fifty, but often noticeably exceed the number of pustules observed in light cases of variola, as many as eight hundred having been counted or estimated; or they may be very few in number (from ten to thirty). They are discrete, or in rare cases two neighboring vesicles may become confluent. Once in a while, on single parts of the body, especially on the extremities, never, so far as I have observed, upon the whole body, there occur unusually small but characteristic groups of varicella vesicles, arranged somewhat in the manner of a zoster, and with no tendency to run together; in the neighborhood of these groups, however, we find, as a rule, plenty of single, large vesicles. They develop usually upon a slightly hyperæmic, and faintly or not at all infiltrated roseola, in such a way that the vesicle finally is surrounded for the most part by a more or less broad areola, though often this is absent. The initiatory hyperæmic spots may interfere with the diagnosis, especially when exceptionally high fever is present; yet even in this stage measles, etc., may be excluded by the dissimilarity of these spots as to size and dissemination, and their smaller number. All doubt is removed, however, by the speedy appearance of small isolated vesicles. The development of these from the initial hyperæmia begins in its centre, and completes itself by peripheral extension, often within a few hours; it is rare that a longer period than a half or an entire day is required.

The fully formed vesicles are clear as water, or light yellow in color, and rise, tightly stretched by their contents, above the level of the skin, like blisters from cantharides or from a burn. They are not umbilicated, doubtless from the rapidity and profuseness of the exudation, and from their superficial situation. When a fresh vesicle is punctured, it gives out a clear, or at most very slightly turbid fluid, at first small in amount, but later comparatively considerable, yet never sufficient to cause complete collapse of the vesicle; it is evidently not a simple vesicular formation, but possesses originally delicate septa subdividing it into single fans, which nevertheless can finally run together in whole or part from the tearing open of their partition walls. Cell layers, lying rather deeper than those which are situated immediately under the horny layer, also doubtless participate, in many cases, in the formation of the vesicles, as is shown by the pigmentations and very faint cicatrices which remain, often only temporarily, but sometimes permanently. Accurate anatomical investigations are impracticable, since varicella is not a fatal disease. The vesicle generally contains a clear, serous fluid, in which are a few pus-cells, and possesses a reaction which is weakly alkaline, according to Hebra, strongly so according to Fuchs and Schönlein, and neutral, according to Canstatt; never, therefore, acid, as in sudamina. In rare cases, when desiccation is unusually protracted, the contents may acquire a somewhat thick, whey-like character, due to moderate increase of the pus-cells; but this, nevertheless, always resembles a very diluted pus, rather than the thick matter of the variola pustules. Apart from this we may find, although the characteristics of the exanthem as a whole are unchanged, here and there a formation of pus, but only in a few tightly stretched, or more frequently flaccid vesicles, which have persisted for an unusually long time; such vesicles are generally found upon the sole of the foot, a place where, on account of the extremely thick epidermis, the pustules of variola also undergo a more protracted involution; they are very rarely found at the same time elsewhere, and the contents are never pure pus. This pus-like condition of the contents of the vesicles, attributable solely to their age, is, moreover, never observed in most cases of varicella, since involution occurs before it can take place. It is, therefore, totally unjustifiable, on account of a few not perfectly clear vesicles, or the not *perfectly* characteristic nature of the exanthem in single cases, and under circumstances which easily explain any deviation from the rule, to at once confound varicella in its totality with the specifically different variola. On the other hand, I must regard those patients who exhibit varicella-like vesicles on particular parts of the body, such as the face, extremities, or back, but at the same time manifest on some of these parts the characteristic pustules of variola scattered among the vesicles, as having variola without the thoroughly typical and universally distributed variola pustules. To admit the possibility of varicella under such circumstances we should have to suppose that the two specifically different diseases were simultaneously present in the same individual, as in the combinations of other acute exanthems, a condition which I have not as yet observed in regard to variola and varicella, but which must be possible, though of course only in children, especially young ones who are unvaccinated. Such a case, according to Trousseau, was published by Delpech in 1845. At times the contents are said to be somewhat bloody, though I have myself never observed it. When air exists under the roof of the vesicle, it has always entered from outside through some aperture (var. ventosæ, emphysematosæ, wind-pox).

The duration of the vesicles is brief. Half a day after their maximum, therefore possibly only one day after the formation of their roseola, they are frequently found, from partial absorption of their contents, flaccid or (though only from external causes, pressure, especially scratching) burst and drying up, their roseola also fading away. Drying, in the case of undisturbed vesicles, begins usually in their centres. They leave behind a small yellowish or clear-brown horny crust, which shrivels gradually and falls off in a few days, leaving the skin either normal or slightly reddened or pigmented; slight cicatrices may also occur, the appearance of which may be very similar to that of a very shallow variola scar. These slight superficial depressions disappear shortly, often leaving no trace, but may, even years later, be in some places so apparent as to give rise to the suspicion that the patient may have had variola, if we simply take into account the existence of the scars, and the circumstance that the patient once had an eruption resembling that of variola. The formation of scars occurs, however, if at all, in varicella in only a few scattered spots (from ten to fifteen at most), or at least it never follows nearly so many vesicles as it does pustules of variola, owing doubtless to the very slight depth and intensity of the affection of the skin; the consideration, therefore, of the number and character of the existing scars has certainly some value in determining subsequently what disease has been present. According to Heim the varicella scar is always white, whiter even than the rest of the skin, perfectly smooth, always hairless, generally round, and feels quite soft, unlike the scar from variola.

The development of the skin affection does not, however, follow this course invariably. I stated that as a rule the formation of the characteristic vesicles of varicella takes place upon slightly or not at all elevated roseolæ, never upon nodular ones. In some very slight cases, however, the majority of these roseolæ varicellosæ simply fade away again, without any appreciable formation of a vesicle taking place, and this is especially apt to be the case also with single roseolæ toward the close of an eruption; or, very imperfect and irregular vesicles of the size of a millet-seed may be formed. In one case, rendered sufficiently certain by the previous characteristic malady of a sister, I even saw all of the numerous roseolæ disappear, after a duration of about thirty-six hours, with no formation of vesicles anywhere. The use of special names for eruptions progressing beyond the formation of roseolæ, but falling short of the fine, tensely stretched vesicles of varicella, is unnecessary.

In contrast with these cases, an unusually intense development of the varicella eruption sometimes occurs. It is frequently observed that small vesicles enlarge somewhat by peripheral growth, while at other times the eventual size may even correspond to the original foundation; the formation in question must, notwithstanding, be still designated as a vesicle. In tolerably rare cases, however, single vesicles attain by peripheral extension such a considerable extent that they become bullæ. These remain for the most part about the size of a dime, but may become as large as, and even larger than, a silver dollar. This expansion of a vesicle into a bulla is most likely to take place when it is still fresh; it may, however, not begin until the crusting of the former has already commenced. In the first case, the bulla possesses the shape of a simple, always more or less flaccid sac; in the second, this sac has a central adhesion from which it stretches outwards in all directions. The epidermis covering the bulla is excessively thin and delicate; I have never seen it tensely stretched by profuse fluid contents, like perhaps the blister of a pemphigus or burn, or like the original vesicle. The epidermis of the periphery becomes gradually more and more raised, and in this way the bulla may still increase even after its roof has been torn and its contents have partially escaped; in fact, it can even make rapid progress in spite of this. How superficial the alterations of the skin are, when bullæ are formed, is shown also by the healing process in these. At times, after the bulla is emptied of fluid, its dry, thin, epidermal covering may be seen lying upon and in connection with the newly formed epidermis, and separating itself only after the completed new formation of the latter. In general the healing process is disturbed by the tearing off of the epidermal roof, and the bulla becomes an open excoriation. In both cases, however, a little crust now forms—always at least in the case of bullæ of any special magnitude,—in the centre of the diseased spot, on the site of the original vesicle, where alone the affection of the skin extends to any depth; and around this, often very speedily, a ring of normal, thin, pale skin appears, already covered again by epidermis. The peripheral margin of this appears at first somewhat excoriated, but at other times it is often in a suppurating condition, the shrivelled and sometimes crusted remains of the roof of the bulla being still attached to it. No marked ulceration, as seen by Hesse and Löwenhardt, ever appeared upon the site of the bulla, and only in the case of small blisters did a thin crust cover the whole basis of the same. There commonly remained upon the site of the bulla a moderate pigmentation of the skin, which decreased only very gradually, while scar-formation, limited always to the site of the central crust, was, after this had fallen, frequently observed. The healing of the bulla was always quite rapid, everything being dried up within a few days. In several very typical cases (all the children in one family) the cicatrices had entirely disappeared at the end of a couple of years.

Symptomatology.

The period of incubation seems to be of more variable length in varicella than in variola or measles. With regard to this I can only adduce observations of cases where the disease spread from one child to another in a single family or house, and where, therefore, it is impossible to specify exactly the moment of infection. If I estimate it, however, from the time of the eruption of the child first affected, I find unusually often that, before the eruption of the next child appears, a period of from thirteen to seventeen days elapses, and we may justly, therefore, consider the period of incubation to be of about this duration, and consequently longer than in variola or measles. The same estimate is made by Trousseau, Körber, Hesse, and others. While the stage of incubation lasts, the general health is, as a rule, perfectly satisfactory, although, as in measles, variola, etc., there may occur, during the whole or any part of this time, slight fever and other general symptoms, such as lassitude, headache, chills, and loss of color.

In most cases the eruption of vesicles is the first symptom which marks the disease. Even very careful and anxious mothers usually notice no prodromal stage, and assert that their children, up to the time of the eruption, have been perfectly healthy. At other times, and likewise by the testimony of non-medical witnesses, an actual precursory stage of some duration appears to have existed. Medical observation has shown that in by far the largest number of cases of varicella, with the usual light, regular, that is to say, normal course, no precursory stage, or, at most, one only of a few hours' duration exists, and it is therefore of no importance. It is truly impossible that in the very first hour of the disease the vesicles should exist fully formed; yet by the precursory stage of an infectious disease is understood a febrile movement of at least one day's duration, without any specific manifestations of disease. Exceptions, of course, exist here also, as in all diseases of children, but under conditions which exclude a normal course of the disease, or which even—where reported by defenders of the identity of variola and varicella, and especially when occurring in children of an advanced age,—permit the suspicion that the case was not at all one of true varicella. And yet, even such as recognize the specific nature of varicella have observed, in very exceptional cases, a fever lasting several days, with severe nervous complications, even delirium and convulsions; the possibility, therefore, of a precursory fever in cases with an abnormal course, cannot be denied. For this very reason, however, special emphasis must be laid upon the fact that the normal course of varicella excludes any prodromal fever, a supposition which is also in some measure justified by this, that the subsequent exanthem in those cases was often altogether disproportionately slight, and frequently even ran its course with a normal temperature, as in perfectly normal and common cases. The possibility exists also that no prodromal fever was present, but merely some febrile complication, casually coincident with the inception of the varicella, and such as often occurs in little children. Careful examinations, made once or twice daily, of the temperature as it existed before the attack during the last days of the incubative stage, furnished the following results:—A precursory stage, manifested by an increase of temperature before the appearance of the eruption, does not in most cases exist; in a few rare cases it can certainly be assumed, but is insignificant, and of only a few hours' duration. Should an essentially more extended precursory stage appear to have existed, upon the authority of non-medical and consequently untrustworthy observations, yet this cannot be regarded as the rule, and will, a little further on, be explained. On the other hand, the positive absence of a prodromal stage of noticeable extent, lasting even half a day for example, can be alleged with great certainty in a number of other cases. I found, for instance, before the eruption, the skin being perfectly healthy, a normal temperature (or very exceptionally, as also frequently occurs in perfectly healthy and uninfected children, a minimal increase, say of 1.8° to 0.54° Fahr.), and noticed, a few hours later, simultaneously with the first increase of temperature, that the eruption had already begun. In these cases, therefore, the fixed beginning of the eruption and of the increase of temperature were coincident, very different from what occurs in variola. Nor has there been found, in the majority of other cases measured from the beginning of the eruption, any more marked initial increase of temperature; on the contrary, this was often on the first day of the eruption normal, or nearly so. This occurred, moreover, not only in cases which ran their course with a normal temperature, or with but a slight increase, but also in such as rose later to, for varicella, a markedly high degree. In only two or three exceptional cases was there any decided initial increase.

The eruption, which is therefore generally the first symptom of the disease to appear, either alone or at least simultaneously with a slight increase of temperature, begins, as a rule, rather irregularly on the upper half of the trunk, and spreads rapidly to the extremities; here, if not upon the first day, certainly by the second, single scattered vesicles may be seen. The face is usually, though not always, the first to be attacked, and the statement, that it in most cases escapes entirely, is incorrect. The hairy scalp shows the exanthem simultaneously; a careful examination will rarely fail to find it here, and then only when the general eruption is very limited. Near the first vesicles faint, red points, of variable size, soon make their appearance, and these often by the next morning will have become in their turn fully developed vesicles, new roseolæ in the meantime having formed between the older eruptions. This always occurs, however, more sparingly upon the head, the part first attacked, and so upon the upper half of the trunk, while the formation upon the extremities is more profuse, and progresses steadily toward the fingers and toes. Soon, in accordance with what has been stated in reference to the brief existence of the individual vesicles, it is only here that we can find any which are characteristic of varicella, those upon the head and trunk having vanished or become scabbed over. It is, in general, only at the commencement of the disease that all the roseolæ develop rapidly into larger or smaller, but always tense varicellæ; towards the end they tend always more and more to simply fade, or produce at best but very small and almost uncharacteristic vesicles. The intensity of the disease seems to diminish, in most cases, with the eruption on the first and second day; that which ensues does not usually manifest the designated type to the same degree. Frequently, by the third day, more often by the fourth or fifth, everything is fading, with the exception perhaps of a few flaccid, discolored vesicles, as large as lentils, upon the hands and feet. More rarely there appears, even at this late period, and in connection with the original affection, a more profuse, fresh eruption on various parts of the body, lasting rarely for more than a day, and never beyond the beginning of the second week after the appearance of the first vesicles.

In accord with this, the duration of the increase of temperature in cases of varicella is a variable one. In about two-thirds of the cases it covers a period of two or three days, in a sixth, of one day, in the remainder, of four days, or more; in a few cases the disease runs its course without any increase of temperature. Except in unusually intense and profuse eruptions this increase of temperature lasts longer than from four to five days only when some complication exists, and in those rather rare cases which lead to a more or less extensive development of bullæ.

Although the appearance of the varicella eruption is only exceptionally accompanied by any considerable increase of temperature, yet as the disease progresses this may reach a degree worthy of regard. The maximum temperature of a case is usually one or two degrees above the normal, but sometimes reaches a noticeably higher point. It was insignificant or normal in only a few very light attacks with moderate eruption. In this also, as in all feverish diseases, there is usually some remission in the morning, and some exacerbation at evening, though this rule is frequently violated by intercurrent elevations coinciding generally with the appearance of numerous fresh vesicles. With any considerable increase of the exanthem, the temperature usually rises, or at least remains at about the height to which it had previously attained, so that, not infrequently, the conclusion of the eruption is marked by the maximal, or certainly by a well-defined rise of temperature. Any frequent coincidence of maximums of temperature and exanthem can hardly be expected in a disease which, properly speaking, shows only in exceptional cases a simultaneous maximum of the exanthem on all parts of the body. The increase of temperature ends by a defervescence, often slight, from the moderate intensity of the fever, but yet always rapid. Any considerable increase of the exanthem, by the production of numerous fresh vesicles, during or at the conclusion of defervescence, has never been observed. A few very small and indistinct vesicles and roseolæ may, even at this late period, make their appearance, possibly because the commencement of their development was of an earlier date. After the expiration of the defervescence no further spread of the exanthem takes place in connection with the development of the principal malady. If new increases of temperature take place, as now and then occurs, they are, in part, ephemeral disturbances of the convalescence, in part due to complications, etc. Generally convalescence is brief, with speedy exfoliation of the little crusts and perfectly normal temperature; there is total absence of any subsequent fever like the suppurative fever in severe forms of variola.

The eruption may be limited to the outbreak of a few small but perfectly typical vesicles, or all parts of the body may be thickly covered by them, and the successive proruptions take place throughout a week. The head and trunk, as also the upper parts of the thigh and arm, are usually covered rather thickly, and tolerably uniformly, with vesicles; the face, however, shows fewer than in variola.

A subsequent eruption of the exanthem is often spoken of in treatises upon varicella. If we understand by "subsequent eruption" the renewed appearance of vesicles after the completed formation and progressing convalescence of the previous eruption, my observation has shown only the appearance of a few, usually not very typical, vesicles, the comprehension of which under the conception varicella admits of some doubt, though their connection with the disease is worthy of regard, and justifies the assumption of their close relation to it. I have seen single vesicles appear a month even after the beginning of the eruption. And yet I cannot admit the propriety of designating the undeniably typical formation of the exanthem—characterized as it is by several rapidly succeeding outbreaks, each of which, after the first, occurs before the previous one has become fully developed, and in which, consequently, are to be found, at the end of the eruption, simultaneously, vesicles in all stages of development and retrogression,—as a primary eruption and a subsequent formation or recurrence. A true relapse, that is, the renewed appearance of the disease in question in its totality, as reported, for example, by Kassowitz, I have never seen. According to Trousseau and Canstatt, relapses in varicella are frequent.

The visible mucous membranes are also attacked by varicella. My attention has frequently been drawn, by their interference with micturition, to vesicles upon the genital mucous membrane of girls, while upon the prepuce of boys they are less common. The mucous membrane of the buccal cavity, and of the throat, is invaded as a rule, though often only moderately. The vesicles are especially evident upon the hard and soft palates, where they may often be seen for a short time as flaccid vesicles upon a somewhat hyperæmic ground, though here later,—as is also the rule elsewhere, as *e.g.*, upon the lips, tongue, and mucous membranes of the cheeks,—they very soon lose their epithelial covering, and are recognizable only as excoriations. A more or less pronounced diffused redness occurs around the vesicles; circumscribed spots like roseolæ are less often seen. The formation of skin upon the spots deprived of their epithelium is always very rapid. I have never seen, as in the case of bullæ upon the skin, any extension or ulcerous development of the spots of mucous membrane attacked by varicella. The conjunctiva I have seen attacked only in continuity with the skin of the eyelids, and in like manner the nasal mucous membrane only at the nasal orifice; both parts, furthermore, being attacked only in rare cases. Henoch has noticed varicella of the conjunctiva and of the gums. I once noticed, just before the outbreak of a light case of varicella with ephemeral though intense fever (106.88° Fahr. in the rectum), the appearance of a universal erythema, of short duration, as with variola and measles. I can assert that I have carefully watched for the appearance of such eruptions, but have never observed any except in this case. The statements of authors, that during a precursory fever lasting several days, erythematous and measle-like eruptions have frequently been present, refer most probably not to true varicella, but to light cases of variola. I have never seen other affections of the skin or of the mucous membranes.

In regard to symptoms on the part of other organs, there is little to be said. In normal, light cases the children are usually perfectly cheerful, or, at the utmost, it is only in the evening, during the exacerbation of the fever, that their general condition is at all disturbed. Slight excitement and restlessness by night, sleepiness perhaps by day, moderate thirst and some loss of appetite, are often the only other signs of disease, and it is only with difficulty that they are kept in bed. Some itching may be present, and temporary difficulty in swallowing, and frequently the lymphatic glands of the throat and neck are somewhat swollen. Headache is usually entirely absent, as is generally the case with young children, nor are there any signs of excessive implication of the nervous system, such as shivering, convulsions, fainting, etc.; vomiting, too, is rare. Symptoms on the part of the thoracic organs may certainly be regarded as complications; and, furthermore, varicellæ of the lining membrane of the respiratory passages are conceivable. If, here and there, in the descriptions of varicella, essentially different and especially more severe symptoms are adduced, it is quite possible that this is due to the fact that the description is based upon the epidemics of other places and other times; as a rule, however, it arises from the confounding of true varicella with an attack of variola which resembles varicella. The distinction between the two cannot be too strongly insisted upon.

Diagnosis.

It cannot be denied that many variola efflorescences, and especially single eruptions not developing into true pustules, have a certain resemblance to varicella. I have never yet been in any doubt about the diagnosis of variola in such cases, especially since the great epidemic of variola in 1871, when my experience with the disease was very extensive. Eruptions resembling varicella in adults always indicate variola, as is shown not only by the age of the patients, but also by a scrupulous investigation of all the symptoms of the disease, and certainty is often eventually established by the character of subsequent cases due to infection from the one first existing. According to my experience it is especially the very light cases of variola, with only a moderate exanthem, occurring in vaccinated adults, and by the mildness of their course resembling varicella, which may give rise to mistakes. The pustules here are scanty and imperfectly developed, and dry up sooner than usual, and instead of pure pus, as in fully developed pustules, their contents are seropurulent. There may even exist one or two pustules with tolerably clear contents, like those of a clouded varicella vesicle, and after the commencement of desiccation a differential diagnosis is difficult. A careful examination will however always detect the nodular formation on the site of the variolous eruption, in contrast with the scarcely swelled basis of the varicella vesicle, and will also discover that it is a question merely with regard to a few suspicious pustules, almost never to a universal eruption on all parts of the body. Furthermore, there never occurs any—I will not say pure, but only approximatively pure—varicellous character in a very profuse exanthem of variola, while even the most intense eruption of varicella is perfectly typical, at least as to the nature of the vesicles. Those vesicles which form frequently around the little pustules in true small-pox, especially in the case of very young children, and which possess clear or light-yellowish contents, and are of course thickly crowded together, so that they form when drying yellow gummy crusts like those of eczema or mellitagra flavescens, can hardly be regarded by any one as a proof of the varicellous nature of the eruption of variola, the characteristic constitution of which is proved by countless other pustules in the same case. If we take the character of the skin alone into consideration, serious difficulties, when sufficient care is exercised, can therefore at most only arise in the case of the most rudimentarily developed forms of the two diseases, those, namely, which barely proceed beyond the formation of roseolæ. The diagnostician, however, encounters similar difficulties, as is well known, in all cases of uncharacteristic disease products. No opinion can be deduced in very old cases from scanty and slightly depressed cicatrices, while profuse and deep scars never result from varicella.

The measurement of the temperature is of great importance for a differential diagnosis. But this must of course be taken during the whole course of the disease, if decisive conclusions are to be deduced from them in a single case; for the difference pertains chiefly to the state of the temperature before the eruption.

In variola this is increased during the precursory stage, which lasts several days, and is marked even in light cases; while in varicella such an increase is entirely absent, or, what is more rare, lasts only a very short time, scarcely more than a few hours. Moreover, in light cases of variola, with which alone we are concerned, the increase of temperature ends with the outbreak of the exanthem, or this last may even not appear until defervescence has commenced. Sometimes, it is true, the elevated temperature lasts for a half or a whole day longer, and defervescence accordingly appears only when the exanthem is already partially developed; at all events, however, its eruption attains its climax with a falling or already normal temperature, and it is often only after the conclusion of the fever that new pocks appear to any extent. Thus the feverish period, even of the mildest variola, with the most scanty exanthem, lasts always several days. It is very different in varicella, in which by the absence of the precursory stage, the period of fever is just so much shortened and the eruption is ended at the conclusion of the defervescence; and when the eruption lasts several days, the temperature, at a later period of the same, tends rather to rise than to become normal. Those who confound both diseases deny positively this typical conduct of the fever for the two affections, although it has been established by accurate observation, and they adduce, in confirmation of their assumptions, single exceptional cases in which this peculiar action has been absent. They forget, in so doing, that the temperature in diseases is never determined from a single point, and that, moreover, in the case of children (especially susceptible as to conditions of temperature) very many and various influences must be regarded. Not a single one of the numerous, recognized, characteristic, typical fever-curves has been constructed from a single case; each is the result of a comparative examination of numerous individual cases. No one can therefore justly make the claim that in every single case all peculiarities of the course of the disease in question must be present in an unequivocal manner. He who desires the characteristic variations of the course of the fever in varicella and variola must take the trouble to prove his assertion on the ground of a careful observation of a large number of pure and uncomplicated cases of both diseases, and must show that, as the result of these observations, the curve of temperature of the two diseases is to be differently constructed from what it is at present; this proof has, however, never yet been furnished by the advocates of the identity of variola and varicella. It must here be emphasized that the typical course of the fever of varicella is best studied, not in the lightest and briefest cases, which run their course with either an ephemeral elevation of temperature or none at all, but in more severe cases, where the eruption lasts at least three days and where no complication exists. Nor, in investigations upon the temperature of the normal course of the fever of varicella, can cases be regarded which evince such manifestations as are, for so mild a disease, unusually severe. When, for example, children with varicella remain for days unconscious, and are attacked by convulsions, and manifest severe nervous or other grave symptoms, it may be justly assumed that their temperature is modified more by this condition than by the light varicella which appears a few days later. In such cases an anomalous condition of the temperature need excite no surprise.

The proofs adduced from the symptoms and course of varicella and variola are truly sufficient to establish categorically the specific differences of the two diseases; we must nevertheless consider a few more important differential points in their pathogeny and etiology.

Vaccination exerts no influence upon a predisposition to varicella. Unvaccinated children who have had varicella can be vaccinated with the best results, and those who have been vaccinated are liable at any time to an attack of varicella. Vaccinia and varicella have therefore nothing in common. The result of the cow-pox inoculation may be perhaps slightly retarded by varicella, as by other diseases, but is otherwise unquestionable, even though vaccination is performed during, or just subsequently to, an attack of varicella. Czakert reports an interesting case where he obtained a successful result from vaccination—in a boy four years of age, who had been three times vaccinated to no effect—by inserting the vaccine lymph into the opened vesicles of a varicella which casually occurred.

Unfortunately he took no lymph from the resulting cow-pox vesicles to establish its infecting properties. Again, the typical course of varicella has never been suppressed, even where its first manifestations have coincided in time with the maximal development of vaccinia. Such cases must be regarded in the same light as those in which, according to existing observations, varicella has developed during the period of florescence of a scarlet fever, or the latter during the progress of a varicella. The case is different with variola. Here everything depends upon the success or failure of the vaccination. Young children who have been vaccinated are rarely attacked by variola, and the susceptibility to this is only subsequently reacquired at the very time in which that to varicella disappears. The advocates of the identity of the two diseases endeavor to diminish the force of this circumstance by a comparison of the percental proportions of the vaccinated and unvaccinated cases of varicella and variola, a demonstration which should be utterly rejected, being based upon false premises. They also make the following assertion: "The mere appearance of vaccinia after varicella, or *vice versâ*, is no reason for not regarding the latter as variola; for it is well known that even variola does not afford us absolute protection against vaccinia, and *vice versâ*—whence the advisability of revaccination. Vaccinia takes after a varicella, but it also takes after a variola, and a limited experience in this respect arises from the fact that vaccination is rarely performed after a patient has gone through an attack of small-pox. With regard, therefore, to the protection by one against the other, it is a mere question of a difference in time, for varicella can immediately follow vaccinia, while variola as a rule can only be contracted after the lapse of a longer, or exceptionally only a shorter space of time, when the soil has, as it were, again become suitable. One can only say: vaccinia is no absolute protection against either variola or varicella, and *vice versâ*. Such facts are, however, insufficient to stamp varicella as a specific disease." In the same way it can of course be demonstrated that, *e.g.*, scarlet fever or typhus is identical with variola, since these afford no protection against vaccinia, and *vice versâ*. The advocates of the identity of the two diseases disregard here the fact that vaccinia does not, as a rule, protect from varicella, while it does, as a rule, protect from variola (and generally, with good lymph, for a long time), and single rare exceptions are not sufficient to overthrow a rule.

The fact that one attack of an acute exanthem almost always destroys any susceptibility to a new attack of the same disease applies as well to varicella as to variola. Since in the former case this predisposition is wont to vanish of itself spontaneously about the eleventh year, literature furnishes only scanty and not always incontestable examples of two attacks of varicella, while such of two, or even more cases of variola, though the latter is rare, are much more frequent. Gerhardt treated a child for three attacks, even, of varicella. Heim reports a similar case. According to Vetter, a child of a colleague was twice attacked, with an interval of fourteen days of perfect health; Kassowitz saw an interval of a year and a half. Boeck has seen a second attack. Hufeland, Canstatt, and Trousseau report repeated attacks in the same child as not infrequent. I have never met with such. If now the contagious principles of variola and varicella, which as yet are unknown, were identical, they ought to afford, at least for a number of years, immunity, each against the other, which is not the case. Unvaccinated varicella patients, who have been treated by the identity-advocates in the small-pox wards of hospitals, are often, immediately after the termination of the varicella, attacked severely by variola; and, on the other hand, there is no lack of cases in which unvaccinated children, soon after a severe attack of variola, go through the most exquisite attacks of varicella. This would occur more frequently, but for the tendency to disappearance of the susceptibility in older children. How do the identity-advocates meet this fact? They allude to the occurrence of single rare cases in which variola also has broken out for a second time, a little while after the termination of a properly diagnosticated case of variola, and take also into account the similar, but less thoroughly confirmed relapses of measles and scarlet fever. They overlook the circumstance that a second attack of variola, following soon after a first, is very rare; while variola following varicella, where so excellent an opportunity is afforded as in the small-pox wards for infection, is the usual condition of things; they also forget that, by such inferences, the identity could be proved of any two diseases whatsoever, for example, of variola and measles (which last disease is also often confounded with variola, so that the patients are sent to the small-pox wards and are naturally attacked subsequently by variola).

The specific nature of varicella necessarily involves the fact that varicella can cause only varicella, just as for the same reason variola can give rise to variola alone. The identity-advocates deny this fact also, and allege in disproof of it that it is quite common for the mildest cases of varicella to give rise to most severe attacks of variola. Such statements are always based upon false diagnosis. If true varicella and a variola resembling varicella are classified under one name, it is self-evident that many a "varicella," which is, in fact, only a misnamed mild case of variola, should give rise to variolous infection. The true varicella of the advocates of non-identity has never given rise by contagion to anything else than another true varicella. We may once more allude to the fact that it is not always possible to make an absolutely certain diagnosis in every case, *e.g.*, where the formation is rudimentary or the eruption already desiccating, etc. Such doubtful cases can never be positively conclusive.

From all these arguments we draw the conclusion that varicella is a specific disease, and has nothing in common with variola. Only the superficial resemblance of the skin affection and the erroneous interpretation of certain facts have led easy-going and reluctant observers to defend the opinion that varicella is the mildest form of variola. This view should be opposed, not only on account of its falsity, but chiefly because it is the principal cause of the totally unjustifiable reproach that the protection afforded by vaccination is entirely problematical. To destroy confidence in vaccination is nothing less than favoring the spread of variola, and forcibly bringing on misfortune which might be prevented. Trustworthy vaccination statistics are only possible when all physicians recognize and rightly diagnosticate varicella as a specific disease.

As the practical result of what has been said, it becomes the duty of every physician to bring about as soon as possible the vaccination of unvaccinated children who have varicella, and never to omit this on account of any diagnostic difficulties whatsoever. For by vaccination in such cases one not only benefits the child, but also at the same time confirms his own diagnosis. Above all things, never expose a case of varicella to an infection by variola, by referring it to small-pox hospitals.

Prognosis.

The prognosis in varicella is thoroughly favorable. The disease, as a rule, runs so mild a course that the physician is not called in, even to young children, and there are weighty impediments to overcome, if one desires to study the malady. Accidents of a severe nature, from intense exacerbations of the fever, are rare; such mishaps, moreover, are usually very temporary, in so far as they are not occasioned by some complication. Grave disturbances of the health, of an indefinite nature, however, have been reported as sequelæ of varicella, but such have never come under my observation. Eczematous, and even ulcerous processes may arise self-evidently from gross neglect of the local disturbance of the skin, especially with that form of varicella in which the small vesicles develop into extensive bullæ.

Complications.

Finally, the combinations of varicella with other acute exanthems deserve special mention. Among these, that with scarlet fever and measles has been repeatedly authenticated in former times (Le Roux, Reuss, Böhm, cited by Hesse) and at a more recent period, and in these cases each disease has been observed to be the first to appear. That varicella can be present simultaneously with vaccinia, has been stated, but whether with true variola has not yet been proved, and would also be difficult probably to prove convincingly. Some (M. Kohn-Kaposi) deny the correctness of the diagnosis of measles or scarlet fever in the above cases, and declare them, on insufficient grounds, cases of variola marked by erythemata or roseolæ, a thoroughly erroneous and untenable supposition.

TREATMENT.

In accordance with the favorable prognosis, the treatment may be, as a rule, purely expectant, and directed, apart from the diet, only to, at most, a few somewhat annoying symptoms, though the affection of the skin may demand a little attention. Any prophylactic isolation of the patient is quite needless, on account of the innocuousness of the malady.

MEASLES.

Compare article "Masern" in the manual and text-book of pathology, etc., by *S. G. Vogel* (2 edit. 1794).—*Conradi* (1813), *Reil* (1815), *Richter* (3. Aufl. 1821), *J. P. Frank* (übers. von *Sobernheim* 1830), *Berndt* (1830), *Puchelt* (1831), *Naumann* (1831), *Eisenmann* (Krkhtsfam. Pyra, 1834), *Baumgärtner* (2. Aufl. 1837), *Schönlein* (5. Aufl. 1841), *Fuchs* (1845), v. *Gaal* (Diagnostik 1846), *Canstatt* (1847), *Wunderlich* (2. Aufl. 1856), *Lebert* (1859), *Leubuscher* (1861), *F. Niemeyer* (5. Aufl. 1863), *Lazansky* (1864), *Trousseau* (1866), *Köhler* (Therapie, 3. Aufl. 1867), *Kunze* (2. Aufl. 1873); in den Werken über Kinderkrankheiten von *Girtanner* (1794), *Rosenstein* (6. Aufl. 1798), *Schäffer* (1803), *Henke* (3. Ausg. 1821, 4. Ausg. 1837), *Capuron-Puchelt* (1821), *Wendt* (1822, 3. Ausg. 1835), *Billard* (1829), *Jörg* (1836), *Fränkel* (1838), *Verson* (1838), *Bressler* (1842), *Schnitzer* und *Wolff* (1843), *Meissner* (3. Aufl. 1844), *Rilliet* und *Barthez* (1844 übers. von *Krupp*, 1856 von *Hagen*), *Friedberg* (1845), *Coley-Hölder* (1847), *Underwood-Schulte* (1848), *Hartmann* (1852), *West-Wegner* (1853), *Bednar* (1856), *Gerhardt* (1861, 2. Aufl. 1871), *Bouchut-Bischoff* (1862), *Hennig* (3. Aufl. 1864), *Rummel* (1866), *Vogel* (5. Aufl. 1871), *West-Henoch* (1872), *Steiner* (1872), *Kormann* (1873); sowie in den Werken über Hautkrankheiten von *Willan-Friese* (1806), *Cazenave* und *Schedel* (1829), *Rayer-Stannius* (1837), *Alibert-Bloest* (1837), *Bateman-Calmann-Blasius* (2. Aufl. 1841), *Simon* (1848), *Fuchs* (1841), *Mayr-Hebra* (1860), *Hebra* (1872), *Neumann* (3. Aufl. 1873). Good catalogues of the literature upon this subject, with especial reference to that of former times, may be preferably found in the works of *Willan*, *Berndt*, *Eisenmann*, *Rayer*, *Canstatt*, *Naumann*, *Barthez* and *Rilliet*, also in *Meissner* (Investigations of the Nineteenth Century, third and sixth parts, Children's Diseases).—See also the Encyclopædia of Medicine, of *Meissner* and *Schmidt* (1832, article On Measles, *Guersent*), also that of *Prosch* and *Ploss* (1855, Art. by *Hennig*).—In the following summary of journal articles and monographs, may be found either such original articles as were accessible to me, or, where these could not be obtained, a designation simply of the sources of which I have availed myself, as I have no wish to immoderately extend the bibliographical table. Discussions and anonymous treatises may be found in the Journal für Kinderkrankheiten 2, p. 74; 4, p. 239; 7, p. 78 und 398; 13, p. 457; 30, p. 172; 40, p. 75 und 77; p. 430; 44, p. 454; in *Schmidt's* Jahrbüchern 1 Supplbd p. 189; 86, p. 237; in *Canstatt's* Jahresber. 1861. IV. p. 204; im Würtemb. Correspbl. 1841 Bd. 11, Nr. 23. 24 und 1861 p. 348; in *Hecker's* Ann. 1829 Bd. 13, p. 428; in *Rust's* Magazin 27, Bd. 1, p. 192.—Single treatises have been written by *Abelin*, Schm. Jb. 94, p. 72.—*Albers*, Schm. Jb. 9, p. 290.—*d'Alves*, J. f. Kdrkh. 9, p. 213.—*Backer*, J. f. Kdrkh. 41, p. 126.—*Bärensprung*, Beob. aus d. med. Gesch. v. Halle 1854.—*Baillie*, u. a. cit. in Dorp. med. Zeitschr. III. p. 205.—*Barbieri*, Cst. Jber. 1864, IV. p. 128.—*Le Barbillier*, Schm. Jb. 92, p. 90. Canstatt's Jahresbericht für 1856. IV. p. 303.—*Bartels*, Virch. Arch. 1861. 21. Bd. p. 65 u. 129.—*Bartscher*, J. f. Kdrkh. 47, p. 28.—*Battersey*, J. f. Kdrkh. 5, p. 339; Cst. Jber. 1845. IV. p. 167.—*Baur*, Würt. Corr. 1861. p. 295, u. 1862, Nr. 37.—*Becquerel*, Schm. Jb. 36, p. 114 u. Pr. Vrtljschr. 82, p. 94.—*Beger*, Cst. Jber. 1860. III. p. 91.—*Behier*, Dorp. m. Z. III. p. 205.—*Behrend*, J. f. Kdrkh. 10, p. 37.—*Behr*, Naum. Pathol. III. 1. p. 690.—*Bentley*, J. f. Kdrkh. 9, p. 155.—*Berton*, J. f. Kdrkh. 1, p. 383.—*Besnier*, Schm. Jb. 140, p. 313.—*Bidault de Villiers*, Alibert I. p. 297.—*Bierbaum*, J. f. Kdrkh. 41, p. 168 u. 178; 42, p. 221.—*Binz*, Jahrb. f. Kdrheilk. N. F. I. p. 223.—*Blanckaert*, V.-H. Jber. 1868. II. p. 256.—*Böning*, Jb. f. Khk. 1871. IV. p. 121; aus D. Klin. 1870. Nr. 30–33.—*Bohn*, Königsb. med. Jahrb. 1859. I. p. 175.—*Bouchut*, Cst. Jber. 1856. III. p. 361, und J. f. Kkh. 39, p. 113.—*Boulay* u. *Caillou*, J. f. Kdrhk. 20, p. 284.—*Bourdillat*, Schm. Jb. 140, p. 67; Med. chir. Rundschau 1868. I. p. 108.—*Brattler*, Beitr. z. Urologie. Schm. Jb. 104, p. 12.—*Braun*, Würt. Corr. 1854. XXIV. p. 237.—*Bresseler*, Cst. Jber. 1841. p. 58.—*Broussais*, Schm. Jb. 62, p. 313.—*Brown*, J. f. Kdrkh. 25, p. 235; Schm. Jb. 88, p. 335; Cst. Jb. 1855. IV. p. 249.—*Brückmann*, Dorp. m. Z. III. p. 205; Meissn. Forsch. III. p. 314.—*Brünniche*, Schm. Jb. 131, p. 298.—*Brunzlow*, Casp. Wochenschr. 1841. Nr. 25.—*Bufalini*, J. f. Kdrkh. 56, p. 282.—*Burserius*, in Berndt's Fieberlehre II. p. 286.—*Carroll*, Oestr. Jahrb. f. Pädiatrik 1870. I. p. 61 An.—*Causit*, Virchow-Hirsch Jber. 1866. II. p. 247.—*Chinnock*, Gers. u. Jul. Mag. 1832. XXIII. p. 133.—*Chomel*, J. f. Kdrkh. 6, p. 126; 8, 375 u. 459; Prager Vtljschr. 15, p. 64.—*Christian*, Centralbl. 1874. p. 95.—*Clarus*, Meissner Forsch. 4, p. 128.—*Clemens*, Cst. Jber. 1850. IV. p. 141; J. f. Kkh. 34, p. 28.—*Coley*, J. f. Kkh. 7, p. 123.—*Constant*, Meissner Kdrkh. III. p. 561.—*Cornaz*, J. f. Kdrkh. 34, p. 302.—*Corrigan*, J. f. Kkh. 5, p. 250.—*Corson*, V.-H. Jb. 1872. II. p. 255.—*Coulson*, J. f. Kkh. 34, p. 440.—*Coze* u. *Feltz*, Schm. Jb. 154, p. 240.—*Cramer*, Pr. Vtljschr. 1 An. p. 113.—*Cullen*, Girtanner, Kdrkh. p. 238.—*Daniell*, Cst. Jb. 1852. IV. p. 210.—*Danis*, Diss. Strasb. 1864.—*Denizet*, V.-H. Jber. 1868. II. 254.—*van Dieren*, Cst. Jb. 1848. IV. p. 142.—*Drake*, Schm. Jb. 90, p. 372.—*Dubini*, Cst. Jber. 1843. Cstt. Spec. Path. p. 219.—*Duchek*. Pr. Vtljschr. 37, p. 95.—*Dumas*, V.-H. Jber. 1872. II. p. 251.—*Duncan*, Dubl. Journ., Sept. 1842.—*Dusével*, Cst. Jb. 1864. IV. p. 129.—*Dyrsen*, Schm. Jb. 28, p. 129.—*Edwards*, Cst. Jb. 1851. IV. p. 146.—*Eiselt*, Dorp. m. Z. III. p. 205.—*Emmert*, Würt. Corr. 1861. XXXI. p. 112 u. 144.—*Engel*, Cst. Jber. 1845. IV. p. 164.—*Erichson*, Bateman-Blasius 2. d. Ausg. p. 80.—*Espinouse*, V.-H. Jb. 1869. II. p. 244.—*Faber*, Würt. Corr. 1852. XXII. p. 221.—*Faye*, J. f. Kkh. 40, p. 233, u. 41, p.

125; Norsk. Mag. 3 R. III. 10. Ges. Verh. p. 126. 1873.—*Feith* und *Schröder van der Kolk*, Schm. Jb. 107, p. 238.—*Fichtbauer*, Würt. Corr. 1841. p. 189.—*Fischer*, Pr. Vtljschr. 24, p. 62.—*Flechner*, Zeitschr. d. Ges. d. Aerzte z. Wien. 8. Jahrg. 1852. 2. Bd. p. 354; Disc. ibid. p. 184.—*Fleischmann*, Jb. f. Kdrhk. 1871. IV. p. 458.—*Flemming*, Arch. d. V. f. wiss. Heilk. III. 1867, p. 200. —*Förster*, Jb. f. Kdrhk. 1868. I. p. 138 u. Pr. Vtljschr. 84, p. 63.—*Fouquier*, J. f. Kkh. 6, p. 66.—*v. Franque*, Wien. med. Presse 1867. VIII. p. 581.—*Fricker*, Würt. Corr. VIII. 1858. p. 390.—*Frölich*, Hufel. Journ. Supplbd. zu 1822.—*Gauster*, Memor. 1869. XIV. p. 270 u. Med. Jahrb. XIX. Jahrg. I. p. 73. 1863.—*Geissler*, Küchenm. Ztschr. f. Med. N. F. I. p. 353. 404. 1862; D. Vtljahrschr. f. öff. Gespfl. III. p. 46.—*Gerhardt*, Jen. Zeitschr. III. p. 118; D. Arch. f. kl. Med. XII. 1. u. 2. H.—*Gierke*, Jb. f. Kdrhk. I. p. 276. Fall 18.—*Girard*, Ctrbl. f. d. med. Wiss. 1865 p. 863; aus Un méd.; V.-H. Jbr. 1869. II. p. 244 u. Arch. f. Dermatol. II. p. 263 aus Gaz. d. Hôp.; Oest. Jb. f. Päd. I. Bd. p. 221. 1870 aus Gaz. méd.—*Gley*, Arch. d. V. f. wiss. Hkde. 1867. III. p. 313.—*Gölis*, Meissn. Forsch. III. p. 315.—*Gottwald*, V.-H. Jbr. 1872. II. p. 255.—*Grätzer*, Kkh. d. Fötus. Breslau, 1837. p. 46.—*Graves*, Dubl. Journ. XVIII. Bd. 1841. p. 238.—*Gregory*, Ausschlagsfieber, übers. von *Helfft* und *Behrend*. 1845. p. 92 ff.; J. f. Kkh. 4, p. 45.—*Gruel*, Würt. Corr. 1862. XXXII. p. 27.—*Günsburg*, Schm. Jb. 68, p. 50; Pr. Vtljschr. 28, p. 86.—*Guersent*, Meissn. u. Schm. Encycl. 9, p. 36: J. f. Kkh. 3, p. 45; Schm. Jb. 6, p. 110.—*Gummers*, Arch. d. Ver. f. wiss. Hkde. II. 1866. p. 486.—*Haartman*, Rosenstein, Kdrkh. p. 323.—*Habisreutinger*, Schweiz. Ztschr. f. Med. 1856. p. 50.—*Hacker*, Schm. Jb. 1 Suppl. p. 196.—*de Haën*, Rat. med. cont. T. 3. p. 352, aus Rosenstein p. 323.—*Häser*, Schm. Jb. 5 Suppl. p. 7.—*Hallier*, Arch. f. Derm. I. p. 51; parasitol. Unters. Leipz. 1868. Centralbl. 1868. p. 161.—*Hannon*, Schm. Jb. 70, p. 65.—*Harnier*, Rust's Mag. XXIII. p. 260.—*Hartmann*, Würt. Corr. 1854, XXIV. p. 200 u. 1862, XXXII. p. 76.—*v. Hauff*, Würt. Corr. 1862. p. 323. 330.—*Hauner*, Jb. f. Kdhk. VI. p. 119–150 u. Ctrlb. 1864. p. 48; Beitr. z. Päd. I. 1863. p. 58 ff.—*Hayden*, J. f. Kkh. 25, p. 424.—*Heberden*, Willan. III. p. 178.—*Hebra*, Hautkkh. 2. Aufl. p. 146.—*Hedrich*, Ztschr. f. Geburtskunde. XV. 3. H.—*Heim*, Hufel. Journ. 1812. 3. St. p. 92.—*Heinecke*, Küch. u. Ploss's Ztschr. VII. p. 188.—*Helfft*, J. f. Kkh. 1, p. 10.—*Henoch*, Beitr. z. Khk. N. F. 1868. p. 366.—*Heslop*, Schm. Jb. 141, p. 298.—*Heyfelder*, Schm. Jb. 24, p. 237; 11, p. 217.—*Hildanus*, Obs. Chir. 56, Cent. IV.—*Hildenbrand* jun., Naumanns Hdb. III. 1, p. 688.—*Hillier*, J. f. Kkh. 39, p. 389.—*Hochmuth*, Diss. Leipzig, 1860.—*Höring*, Würt. Corr. 1841, p. 186; 1856, p. 304; 1857, p. 183.—*Hofmann*, Ztschr. f. Paras. III. 1872. p. 105.—*Holmes Coote*, Schm. Jb. 124, p. 303.—*Home*, Rosenstein Kdrkh. 6. Aufl. p. 353.—*Hood*, J. f. Kdrkh. 5, p. 264.—*Huguenin*, pathol. Beitr. Zürich, 1869.—*Huxham*, Reil's Fieberl. V. p. 225.—*Imbert-Gourbeyre*, Cst. Jb. 1857. IV. p. 223.—*Jaccoud*, V.-H. Jbr. 1871. II. p. 250. —*Jahn*, Entexantheme. p. 42—*Jenner*, J. f. Kkh. 55, p. 12.—*Jorritsma*, Gerson u. Julius Mag. 1830. Bd. 19 p. 1.—*Jütting*, J. f. Kdrkh. 3. p. 250.—*Just*, Cst.

Jber. 1860. III. p. 91.—*Kapff*, Würt. Corr. 1838. 8. Bd. p. 13.—*Karajan*, Med. Jahrb. XXI. Jhrg. 2. Bd. p. 17.—*Karg*, Woch. d. Wien. Aerzte. 1866. Nr. 49. 50; 1870, Nr. 35. 36. 37.—*Kassowitz*, Jb. f. Khkde. N. F. VI. p. 165.—*Katona*, Oest. med. Woch. 1842. Nr. 29.—*Kaufmann*, Bair. Intell. 1862. Nr. 20.—*Kaurin*, V.-H. Jbr. 1868. II. p. 252.—*de Keghel*, V.-H., Jbr. 1869. II. p. 245.—*Kellner*, Frankf. Jber. f. 1858. II. p. 36.—*Kennedy*, J. f. Kkh. 25, p. 417.—*Kerschensteiner*, Jb. f. Kdhk. 1858. I. p. 54, Anal. oder Pr. Vtljschr. 57, p. 68.—*Kesteven*, Schm. Jb. 135, p. 239.—*Kierski*, Berl. kl. Woch. 1868. p. 326.—*Kjellberg*, J. f. Kkh. 54, p. 193.—*Koch*, Würt. Corr. 1863. Bd. 33. p. 255.—*Köhler*, Würt. Corr. 1841. p. 190.—*Köstlin*, Arch. d. V. f. wiss. Heilk. II. 1866. p. 338; Würt. Corr. 1856. p. 31 und 1862, p. 197.—*Kohn*, Wien. med. Woch. 1867. Nr. 41-43.—*Kolb*, Würt. Corr. 1841. p. 190.—*Krieg*, Cst. Jbr. 1843. p. 219. Spec. Path.—*Kronenberg*, J. f. Kdrkh. 4, p. 243.—*Krug*, *C. A.*, Diss. Leipzig, 1841.—*Küttner*, J. f. Kkh. 30, p. 182.—*Lafaye*, V.-H. Jber. 1868. II. p. 257.—*Laveran*, Schm. Jb. 118, p. 184; Pr. Vtljschr. 71, p. 67; Cst. Jbr. 1861. IV. 205.—*Ledelius*, Ephemerid. Dec. II. Ann. III. 1684. Observ. 97, p. 204.—*Lees*, Pr. Vtljschr. 8, p. 50; J. f. Kkh. 3, p. 333.—*Lehmann*, Schm. Jb. 139, p. 240.—*Levy*, Schm. Jb. 62, p. 311; Cst. Jbr. 1847. IV. p. 99.—*Lewin*, J. f. Kkh. 42, p. 95.—*Lievin*, D. Vtljschr. f. öff. Gespfl. III. p. 358. 362 ff.—*Liverani*, J. f. Kkh. 56, p. 291.—*Löschner*, Pr. Vtljschr. 3, p. 37; Prag. med. Woch. Nr. 24, p. 192. 1864; Epidem. u. klin. Stud. Prag, 1868. p. 26; Jbch. f. Khk. 1865. VII. 1. H., p. 12 und 3. H., p. 45. 57; Jbch. f. Kdhk. 1868. I. 468.—*Luithlen*, Würt. Corr. 1854. XXIV. p. 207.—*Luzsinsky*, J. f. Kdrhk. 32, p. 206.—*Majer*, J. f. Kkh. 56, p. 161.—*Malmsten*, V.-H. Jbr. 1869. II. p. 246.—*Marsden*, J. f. Kkh. 12. p. 301.—*Martineau*, V.-H. Jbr. 1866. II. p. 246.—*Masarei*, Oest. med. Jahrb. Bd. 63. 1848. I. p. 173.—*Mason*, Schm. Jb. 155, p. 68.—*Mauger*, V.-H. Jber. 1866. I. p. 237.—*Mauthner*, J. f. Kkr. 12, p. 447; 21, p. 289.—*Mayo*, Cst. Jbr. 1844. III. p. 229.—*Mayr*, Wien. Ztschr. 1852. VIII. I. 6. 97. p. 193. II. Bd. p. 193.—*Mertens*, Cst. Jbr. 1852. IV. p. 210.—*Mettenheimer*, Arch. d. Ver. f. wiss. Hkde. III. 1867. p. 343; J. f. Kkh. 1872. 58. Bd. p. 1.—*Meyer-Ahrens*, Pr. Vtljschr. 54, p. 144.—*Meyer-Hoffmeister*, Schweiz. Ztschr. f. Med. 1849. p. 468.—*Michaelsen*, Schm. Jb. 8, p. 291.—*Michele*, Cst. Jbr. 1861. IV. p. 204.—*Mombert*, Schm. Jb. 2. Suppl. p. 153.—*Monti*, Jb. f. Khk. 1864. I. p. 52; Ibid. 1865. VII. p. 52; 1869, N. F. II. p. 77; 1873, VI. p. 20.—*Moore*, Schm. Jb. 3 Suppl. p. 457; J. f. Kkh. 17, p. 363.—*Müller*, Schm. Jb. 72, p. 271.—*Müller*, R. L., Diss. Leipzig, 1870.—*Müller*, Jen. Ztschr. f. Med. IV. p. 167. 1868. *Mugnier*, Schm. Jb. 136, p. 140.—*Naunyn*, Berl. kl. Woch. 1868. p. 237.—*Neisser*, J. f. Kkh. 3, p. 196.—*Neumann*, V.-H. Jbr. 1869. II. p. 244.—*Neureutter*, Oest. Jb. f. Päd. 1871. II. p. 115.—*Nicola*, Beob. u. Abh. öst. Aerzte. II. Wien, 1821. p. 361.—*Nicolai*, Schm. Jb. 10, p. 156.—*Obermeier*, Virch. Arch. 54, p. 545.—*Oesterlen*, Cst. Jber. 1841-42. II. p. 13.—*Ottoni*, Cst. Jber. 1861. IV. p. 204.—*Osiander*, Stark's Arch. f. d. Gebh. 5. Bd. 3. St. Jena, 1794. p. 572; Hdb. d. Entbdgsk. I. p. 596.—*Pank*, J. f. Kkh. 4, p. 379.—

Panum, Virch. Arch. I. 1848 und Würzb. Verh. 1851. II. p. 291.—*Passow*, V.-H. Jber. 1869. I. p. 285; D. Vtljschr. f. öff. Ges. II. p. 158.—*Pastau*, Petechialtyphusepid. in Breslau. Br. 1871. p. 23 u 24.—*Pfeilsticker*, Beitr. z. Pathol. d. Mas. Tüb. 1863.—*Pinkham*, V.-H. Jber. 1871. II. p. 251.—*Pockels*, Varges Ztschr. 14, p. 1.—*Polak*, Pr. Vtljschr. 28, p. 19.—*Politzer*, Jbch. f. Khkde. 1871. IV. p. 307.—*Posner*, J. f. Kkh. 7, p. 175.—*Ranke*, Jb. f. Kdhk. 1869. II. p. 34.—*Rautenberg*, J. f. Kkh. 56, p. 41. 46. 55.—*Ravn* u. *Aarestrup*, V.-H. Jber. 1867. II. p. 6.—*Rehn*, Jb. f. Khk. 1868. I. p. 93.—*Reichard*, Würt. Corr. 1841. p. 186.—*Reuss*, Hufel. Journ. Suppl. zu 1822.—*Richter*, Schm. Jb. 151, p. 340.—*Riecke*, Schm. Jb. 28, p. 133.—*Rilliet*, J. f. Kkh. 10, p. 359 u. 432.—*la Rivière*, Journ. de Bord. 3. Sér. I. 1866. 15. Janv.—*Röhling*, Diss. Leipzig, 1870.—*Rösch*, Würt. Corr. 1843. p. 180.—*Röser*, Würt. Corr. 1854, p. 209 und 1855. p. 35.—*Roger*, Journ. f. Kkh. 4, p. 66 u. 257.—*Roncati*, J. f. Kkh. 55, p. 447.—*de Roseville*, Cst. Jber. 1862. IV. p. 133.—*Rota*, J. f. Kkh. 47, p. 346; V.-H. Jbr. 1866. II. p. 248.—*Rothe*, Berl. kl. Woch. 1870. VII. p. 292.—*Routh*, J. f. Kkh. 27, p. 314 und 28, p. 419.—*Roux*, V.-H. Jber. 1869. II. p. 245.—*Rüttel*, Canstatt Pathol. 1. Aufl. II. p. 188 Anm.—*Rufz*, Cst. Jbr. 1857. IV. p. 222.—*Russell*, Schm. Jb. 155, p. 87.—*Sadler*, J. f. Kkh. 42, p. 122.—*Salzmann*, Würt. Corr. 1860, p. 60 u. 1862, p. 154.—*Schallenmüller*, Würt. Corr. 1838. p. 310.—*Schepers*, Berl. kl. Woch. 1872. Nr. 43.—*Schiefferdecker*, Einfl. d. acut. Hautausschl. auf d. Kdrstrblchkt. 1870.—*Schneemann*, Cst. Jber. 1853. IV. p. 311.—*Schott*, Würt. Corr. 1848. p. 79.—*Schreiber*, Wien. med. Presse 1865. VI. p. 929.—*Schüz*, Würt. Corr. 1858. p. 37.—*Schultze*, Arch. d. V. f. wiss. Hkde. 1867. III. p. 204 u. 416.—*Schwartze*, J. f. Kkh. 42, p. 157.—*Schwarz*, Wien. m. Presse 1868. IX. p. 302 u. 323.—*Scoutetten*, Pr. Vtljschr. 69, p. 82; Cst. Jber. 1859. IV. p. 124.—*Seidl*, Oest. med. Jb. 1843. Bd. 45. p. 263.—*Seitz*, Bayr. Intell. 1873. p. 756 u. Pr. Vtljschr. 5, p. 85.—*Sibergundi*, Naum. Hdb. III. 1. p. 688.—*Siegel*, Arch. d. Heilk. 1861. II. p. 521.—*Simpson*, J. f. Kind. 26, p. 124.—*Smoler*, Pr. Vtljschr. 82, p. 94.—*Spiess*, Frankf. Jahresber. f. 1867. XI. p. 40.—*Steinbacher*, Scharl. u. Mas. Augsb. 1865. p. 183.—*Steiner* u. *Neureutter*, Pr. Vtljschr. 84, p. 92 u. 106, p. 73.—*Steinthal*, J. f. Kkh. 42, p. 333.—*Steudel*, Würt. Corr. 1841. p. 183.—*Stiebel*, Schm. Jb. 94, p. 72; J. f. Kkh. 28, p. 64; ibid. 33, p. 146.—*Stilling*, Arch. d. V. f. wiss. Hkde. 1866. II. p. 139.—*Stoffella*, Wochbl. d. Wien. Aerzte 1862. p. 154.—*Taupin*, J. f. Kkh. 1, p. 379.—*Thaer*, Heck. Ann. 1829. Jan. XIII. p. 19.—*Tholander*, V.-H. Jber. 1869. II. p. 246.—*Thomas*, Arch. d. Heilk. 1867. VIII. p. 370. 385.—*Thomas*, Thèse de Paris; V.-H. Jber. 1869. p. 245.—*Thore*, Schm. Jb. 118, p. 184; Cst. Jb. 1861. IV. p. 205.—*Thoresen*, V.-H. Jb. 1869. I. p. 219.—*Thuessink*, Meissn. Forsch. VI. p. 450.—*Tourdes*, J. f. Kkh. 23, p. 125.—*Tresling*, V.-H. Jber. 1872. II. p. 256.—*Triboulet*, J. f. Kkh. 48, p. 435.—*Trojanowsky*, Dorp. m. Z. 1873. III. p. 199; IV. p. 19.—*Trousseau*, J. f. Kkh. 3, p. 239 u. 317; 15, p. 429; 21, p. 123; 35, p. 398 u. Schm. Jb. 104, p. 191; Cst. Jber. 1852. IV. p. 203.—*Tüngel*, Klin. Mitth. f. 1860. Hamb., 1862. p. 19.—*Tufnell*, Jb. f. Kdrhk. 1873. N. F. VI. p. 186; V.-H. Jb. 1872. II.

p. 255.—*Veit*, Berl. kl. Woch. 1868. . p. 452 ff.; Virch. Arch. 1858. XIV. p. 64.—*Vogel*, Arch. f. Derm. 1870. II. p. 408; J. f. Kkh. 19, p. 109.—*Voit*, Jb. f. Kdhk. 1872. N. F. V. p. 256.—*Volz*, Häser's Arch. VI. p. 316. 1844.—*Walz*, Prager, Vtljschr. 40, p. 72; Cst. Jber. 1853. IV. p. 169.—*Weber*, Varges Zeitschr. 11, p. 36.—*Weber*, *H.*, Schm. Jb. 133, p. 332; Arch. d. V. f. wiss. Hkd. 1866. II. p. 358.—*Webster*, Gregory, Ausschlagsf. p. 103.—*Weil*, Schweiz. Ztschr. f. Med. 1850. p. 275.—*Weineck*, Die Epidem. d. Stadt Halle. Halle, 1872.—*Weisse*, Schm. Jb. 18, p. 375; J. f. Kkh. 9, p. 368; 27, p. 52.—*West*, J. f. Kkh. 12, p. 117; 39, p. 408; Pr. Vtljschr. 24, p. 24.—*Williams*, Schm. Jb. 21, p. 141.—*Wilson*, Berndt Fieberl. II. p. 286; Heck. Ann. 1828. Nov. XII. p. 353; Jahn Entexantheme, p. 42; Jbch. f. Kdhk. 1858. I. p. 46.—*Wisshaupt*, Pr. Vtljschr. 22, p. 101.—*Wolf*, Arch. d. V. f. w. Heilk. 1864. I. p. 433.—*Wood*, Centrbl. 1868, p. 841.—*Woodward*, Schm. Jb. 124, p. 121.—*Wunderlich*, Arch. f. phys. Heilk. 1858. XVII. p. 14; Arch. d. Heilk. 1863. IV. p. 331; Eigenwärme in Kkh. II. Aufl. Leipz., 1870.—*Wunderlich* und *Weil*, Arch. d. V. f. wiss. Hkd. 1867. III. p. 196.—*Zavizianos*, V.-H. Jber. 1866. II. 245.—*Zehnder*, Schw. Ztschr. f. Hkde. 1863. II. p. 425.—*Ziemssen*, Greifsw. med. Beitr. I. p. 117.

HISTORY.

MEASLES were certainly recognized in old times. We need have no hesitation, according to Hirsch, in pronouncing as measles the eruptive disease described by Rhazes under the name of Hhasbah, and there is no doubt that many observers in all parts of the world during the fourteenth and sixteenth centuries were acquainted with measles and described it under the name of morbilli or blacciæ. At the same time its specific nature was mostly unknown. Many other acute macular exanthemata were confounded with true measles, and a true portrait of it is therefore rarely recognizable in the descriptions. The first indisputable records of true epidemics of measles are furnished, according to Fuchs, by Forest (1563), Lange (1565), Ballonius (1574–5), and Schenk (1600); accurate knowledge of these, however, has been more especially furnished by Sydenham and Morton (1670–74), though even they have not distinguished it with sufficient accuracy from scarlet fever. Only since the middle of the last century, however, has measles been regarded as an unquestionably specific and independent disease.

Where measles originated, when and how it spread, and to what extent it has prevailed of old in the inhabited portions of the earth, are questions which do not admit of even a tolerably grounded hypothesis. Probably it is of exotic origin.

ETIOLOGY.

Daily experience teaches that measles is contagious. It needs, as a rule, only about a week, when the disease has been introduced by a patient into a family, or body of persons living together, for those among them who are predisposed to it to become infected. The usually rapid increase of such individual foci occasions an epidemic, the extent of which depends, in general, upon the number of those exposed who have not already had the disease. The contagiousness is furthermore somewhat shown by the positive results of inoculation. On the other hand, it is not likely that measles nowadays develop spontaneously. There exist, at any rate, large tracts of country never as yet visited by epidemics of measles, doubtless because the contagion has not yet reached them (Australia); while others under the most varying conditions of climate have been spared either completely, or for long periods, and then finally have been visited by the disease. If now and then cases have been related where persons, for a long time removed from all intercourse, have fallen ill of the measles, yet this circumstance by no means proves that this has arisen spontaneously, and not been communicated by any infected person. In many cases the infection, which beyond a doubt has occurred, cannot be pointed out; but this proves nothing, since where a contagion is so volatile and diffusible we must expect that it will attach itself or become adherent to material objects. In the case of contagious diseases it must never be asserted as impossible that the infecting material has been introduced by means of clothing, furniture, or even the physician himself (especially after a long stay in a sick-chamber). It may be mentioned in this connection that Mayr believes himself justified in denying any excessive tenacity of the contagion, since he could not ascertain that the disease was communicated by the garments and underclothing of patients brought into the hospital with measles, when such clothes, after being aired, were made use of for other children in the building. All these questions cannot, of course, be decided with absolute certainty before we have acquired an accurate knowledge of the nature of the contagion, its mode of extension, and the conditions of its existence and activity. We can, at all events, reject absolutely the old hypothesis, according to which measles was to be regarded as a catarrhal process, peculiarly modified by atmospheric influences solely, and indicating a highly developed catarrhal constitution in the person affected.

The nature of the contagious principle of measles is still entirely unknown. From the results—which are by no means free from suspicion—of inoculations, the propriety has been inferred of regarding it as present in the blood, especially that of the measle-spots upon the skin, in the contents of the miliary vesicles which develop upon these spots, in the tears, and the secretion of the nasal mucous membrane, perhaps also in the saliva and in the sputa. A peculiar smell was ascribed to it by Heim and other older authorities.

Direct examinations of the blood and the secretions have thus far furnished thoroughly contradictory results. Hallier found in the sputa, and also in the blood of measles patients a tolerably large number of free cocci, mostly with a tail-like end, movable and colorless, smaller than those in typhus; single blood-corpuscles only were infested or filled with micrococci. These fructified, in his attempts at cultivation, upon various substances or fluids, and produced always one and the same fungus (mucor mucedo verus Fres.). Vogel contests most decidedly the possibility of proving these statements. Salisbury ascribed measles to a fungus-spore found upon decaying straw; inoculations with this parasite were alleged to produce an exanthem bearing a close resemblance to measles. He asserts that he has also found that several people, in the neighborhood of those upon whom measles had thus been, *experimenti causa*, generated, manifested the same disease after a period of incubation of from one to two weeks; while the eruption showed itself after the lapse of from thirty-four to seventy hours in the cases of those who had handled the decayed straw. On the other hand, Pepper inoculated twenty-two persons, who had not yet had measles, with this fungus, without any result. Coze and Feltz found in the blood of measles patients numerous bacteria of great activity and extreme delicacy. They noticed also that, in man, the blood which was richest in bacteria came from those spots where the exanthem was most pronounced. The nasal mucus contained, in the stage of invasion, numerous similar elements. The inoculation of the blood of measles upon rabbits produced, however, a fever of two or three days' duration, with speedy recovery, and clearly of a very different nature from the measles of man. The attempts, therefore, to demonstrate the contagious principle of measles, have thus far proved unsuccessful.

The successful inoculation of measles, by means of the blood as a vehicle, was first attempted in 1758, in Edinburgh, by Home, at the instigation of Monro. He laid for three days, upon fresh cuts in the upper arm of a healthy person, rags soaked in blood which had been taken from cuts made through the spots of measles, upon the day of the disappearance of the disease. The resulting affection was very mild, differing essentially from the severe type prevailing at the time. The rags soaked in blood retained their infecting properties only ten days. Willan states that he once inoculated the fluid of a miliary vesicle of measles without success, while Wachsel, on the contrary, was successful. Subsequently the tears of measles patients were employed, by moistening lint with them and laying it upon slight incisions in the arm of the subject of the experiment; saliva also, and the branny desquamation of the skin, have been made use of. Themmen tried inoculation in 1816, at von Thuessink's suggestion, but with no result; it was tried in Philadelphia in 1801, tears, blood, mucus, and epidermis being employed; also by Albers, who doubted the credibility of Home's experiments, because these had been made in a hospital continuously occupied by patients with measles, and the infection might therefore have arisen in some other way. Speranza, however, in 1822, confirmed the experience of Home; he himself was inoculated most successfully. Bufalini likewise made in Italy, in 1854, successful experiments in the inoculation of measles, and reports similar experiences on the part of his countrymen Locatelli, Rossi, Figueri, also of Horst and Percival. In 1842 Katona published numerous (1,122) inoculations, which mostly succeeded and produced throughout only a mild disease, whilst otherwise a severe epidemic was prevailing. No inoculated person died, and only seven per cent. of the inoculations failed. The inoculations were made chiefly with a mixture of blood and of the contents of miliary vesicles, sometimes with tears, in the same manner as vaccination. Around the point of incision was formed a red areola, which gradually disappeared, as did also the mark of the incision; and the prodromes, which were ushered in by fever on the seventh day after the inoculation, were followed by the appearance of the eruption of measles on the ninth, or at the latest tenth day after inoculation, the disease then running a customary though exceedingly mild course. On the fourteenth day the fever usually disappeared, and on the seventeenth the patients might be regarded as well. Mayr inoculated successfully in 1848 and in 1852. In opposition to these observations, Jörg asserts that measles from inoculation ran in no case any lighter course, and declares these therefore as useless; Wendt is of the same opinion. The question therefore of the inoculation of measles cannot yet be regarded as sufficiently elucidated.

That the nasal mucus carries the contagion, has been shown by Mayr. At the close of an epidemic of measles in the city, he placed freshly secreted mucus from the nose, and some which had been preserved in a glass tube, upon the mucous membranes of two children. The first signs of catarrh manifested themselves, in the former case, after eight, in the latter, after nine days, and two days later fever followed. The outbreak of an eruption of measles succeeded, in both cases, on the thirteenth day after infection occurred, and the disease ran a regular course.

On the other hand, the attempts made by Mayr, with epidermis scales from children in the stage of desquamation, failed, as had already before happened to Monro. Yet Berndt asserts that Monro and Look inoculated successfully the desquamation, the tears, and the saliva of measles patients. In this respect the exfoliative product of measles differs essentially from the crusts of variola, which readily communicate this disease. Cullen states that children have eaten the scales of measles without detriment, as Girtanner relates.

Every object which has in any way come into contact with infected persons, or has been in their atmosphere, may serve as a vehicle of contagion. Even a healthy physician can, by means of his clothes, or of any other thing he carries with him, occasion infection, as in several instructive cases mentioned by Panum. So, for instance, measles broke out in a house which had had no intercourse with the rest of the world, except that a physician had spent the night there a fortnight before, he having come from an infected district four miles away, and been compelled, moreover, to travel in an open boat, in stormy, rainy weather ; in the same way, it is alleged, has the disease been introduced into uninfected houses by a midwife just recovered from the measles. Other observations seem to show that contagion does not necessarily follow when the contact with the infected clothes, etc., has been brief. Thoresen asserts that transportation by a healthy person rarely takes place, since the contagion of measles does not readily adhere to clothes, it being very different in this respect from that of scarlet fever. Thuessink assures us that he knew of a case where the infection was occasioned by a letter; and another where it was attributed to an engraving sent per post. How long the contagion in clothes and other objects remains active, is not known; its tenacity appears, however, to be but moderate.

In most cases contagion is due to association with those infected, and a slight exposure is often sufficient to superinduce the disease, while in other cases a prolonged one is needed. In this connection we should bear in mind not merely the susceptibility of the persons infected, but also the total amount of contagion prevailing in the district of infection. Where apartments are insufficiently ventilated, or crowded with patients, the contagious principle must be present in greater amount than under opposite circumstances, and a slight contact will then suffice to communicate the virus to one susceptible to its influence. The necessity for suitable hygienic conditions speaks for itself.

By far the most frequent opportunity for infection, with the exception of family intercourse, in which the danger is proportionate to the number of occupants of the house (Geissler) or dwelling-place, is afforded by the "Kindergärten" and the schools (Spiess, Gruel, Veit). Here large numbers of children from the most different parts of the city are brought into close contact, furnishing the most advantageous field for infection, by the propagation of which, from a limited number of foci, throughout the entire district, they serve as the actual cause of a rapid spread of an epidemic. The children infected at school introduce the disease into the entire home circle down to the youngest infant.

Susceptibility to the contagion of measles is almost universal, except where one attack has already occurred. Second attacks of measles are, according to the experience of most observers, exceedingly rare, as much so as second attacks of variola, scarlet fever, varicella, etc. Third attacks are even less frequent, though their occurrence, as in cases of variola, cannot be doubted. Thus Van Dieren reports the case of a girl, three years of age, who was attacked by measles in the beginning of February, 1848, and recovered; but was again attacked on March 4th, most characteristically, with the preliminary stage and the exanthem; and the whole process, accompanied in the precursory stage by vomiting and convulsions, began again on the 12th of April, each of these three attacks being succeeded by a branny desquamation. The cases, however, reported by Dyrsen, Bierbaum, Spiess, and Home, do not exclude the possibility of deception. Nor can we regard as incontestable all the cases of second attacks which are recorded. In regard to these it is at all events suspicious that the report is frequently made only with reference to single epidemics, or even to the circle of observation of single physicians during an epidemic, that a more or less frequent occurrence of second attacks of measles has existed; nay, at times it is only one and the same observer who relates that he has seen recurrences in the one epidemic of his dwelling-place, but not in others. So, for example, Spiess reports that the Frankfort epidemic of 1866–67 was characterized by unusually frequent recurrences, individual physicians having testified that the half, at least, of their cases had been relapses from this or a previous epidemic. This excites the well-grounded suspicion that rubeola,—since there is no doubt of its existence as an independent disease,—on account of its resemblance to measles, may have been frequently confounded with true measles; and this is all the more likely if the defenders of the frequency of relapses in measles deny the specific nature of rubeola. Apart from rubeola, the simple non-specific roseola may have often given rise to confusion. Against the frequent and even customary recurrence of measles, especially espoused in recent times by Trojanowsky, we possess the strongest evidence of good authorities; I mention only the names of Berndt, Thuessink, Schönlein, Mayr, also Schott, and Bartscher, with whose experience my own thoroughly coincides. Even Panum, from his immense number of observations upon people of every age, has not been able to report a single case of a second attack of measles. The experienced Rosenstein testifies that he has for forty-four years, and Willan, that he has for more than twenty years, devoted the closest attention to eruptive diseases, without ever meeting with any one who had had for the second time measles accompanied by fever; and that there are therefore good grounds for fearing that some error has occurred in the cases apparently contradicting such an experience. Stiebel alone asserts that according to his experience many, nay, most persons, are susceptible to a second attack of measles; we seek in vain, however, for more definite statements in his accounts.

Cases of a second attack of measles may be divided into such as appear a long time after the first attack,—that is, at least a quarter or half a year, or generally much later, even from twelve to eighteen years afterwards,—and into such as occur as early even as a few days after the first attack, or, at most, from three to four weeks afterwards. Cases of the first sort are reported by Battersey, Roberdière, Rayer, Flemming, Kassowitz, Haartman, Webster, de Haën, Stiebel, Brunzlow, Luithlen, Mauthner, Kierulf, Trojanowsky, Home, Baillie, Lewin, Gauster, Karg, Béhier, Tresling, Spiess; here, if the diagnosis was correct, there is no doubt of a second infection. Bierbaum relates a case in which measles appeared three times before the thirteenth year. On the other hand, a subsequent infection in cases of the second sort is less certain; they should rather be regarded as mere relapses, and be estimated in the same way as the well-known relapses of typhoid fever. It seems to me improbable that these typhoid relapses owe their origin to a subsequent infection from without, and Gerhardt calls attention in this connection to the duration of the swelling of the spleen after the conclusion of the fever; the supposition seems to me more credible, therefore, for measles also, that some internal cause for the new disease is in very rare cases produced by the first attack. In favor of this is the circumstance that the free interval is usually of a tolerably definite duration. Löschner reports observations, according to which, after the conclusion of the measles and a perfect recovery, the disease began again, and once more ran through all its stages. Such cases are also related by Spiess, Mettenheimer, Gauster, Vézien, Wilson, Koch, Barbillier, Graves, Eiselt, Schüz, Köstlin, Chinnock, Riecke (?), Brückmann, Böttiger, Behr, Schultze, Abelin, Faye, Bidenkap, Nicola, Kierulf, Rüttel, Thaulow. According to v. Düben and Malmsten, the interval can amount to only a few days. Spiess, on the other hand, reports an interval of from one to two months; so also Stilling and Wendt (according to S. G. Vogel), one of six weeks; Steiner and Wunderlich, one of eight weeks; so also Stiebel, Bresseler, and others. Lippe, who, in the course of three epidemics, has seen fifteen cases of this sort, remarks that the children concerned were newly attacked, three or four weeks after the first disease, by coming into close contact with a child lying ill with its first attack of measles; these second attacks were, as a rule, more severe the milder the course of the former had been. The same was observed by Seidl, who three times saw a violent and malignant recurrence from four to six weeks after a first mild attack, and in two of the cases death followed. If future observations should prove the free interval as in fact of a nearly definite duration, it can then be assumed as in the highest degree probable that a definite relation exists between the original disease and the relapse, and not the casual one of reinfection. Thus Rufz reports cases where, in the first attack, the exanthem did not break out, and, as in morbilli sine morbillis, only catarrhal symptoms existed, while the eruption was perfect in the second attack, which appeared two or three weeks later.

Whenever the exanthem of measles, in the first attack, is not universal, or is merely rudimentary in development, relapses seem to be more frequent than when there has been an intense eruption.

There may also be some justification, in the explanation of single cases of true recurrence of measles, for Meissner's supposition, that it depends upon a temporary suppression (metastasis) and a subsequent reappearance of the morbid process. Thus Brückmann observed a boy who, after a normal course of measles, was attacked by quite a violent suffocative catarrh, which lasted over four weeks, and only disappeared after a second outbreak of measles, which ran a normal course. Rosenstein attributed such cases to a swollen gland remaining from the first eruption, and engendering somewhat later a fresh outbreak. Trojanowsky believes that subsequent attacks, occurring after the lapse of years, may often be explained by geographical differences in the contagious principles causing the attacks.

The so-called "recurrent form" described by Trojanowsky occupies a different position, both as regards its course and its prognosis. The most typical factor of this is not the exanthem, but the fever, which closely resembles relapsing fever, though not in every respect identical with it. It appears in the form of two, generally pretty violent, paroxysms, which, as in the case of relapsing fever, rapidly supervene and tolerably quickly disappear again, possess an average duration of from six to eight days, and are separated by an interval of normal temperature lasting about eight days. The highest elevation of temperature (105.5° Fahr., and even higher) shows itself on the second and third days of the disease, simultaneously with the highest development of the exanthem, which closely resembles that of measles, but whether identical with it I am unable to state, at least from personal experience; at all events an affection of the conjunctiva, and of the mucous membrane of the air-passages, of the nature of measles, certainly does exist. The distinctive factors of such an attack are the violent fever, the premature eruption and florescence of the exanthem, as well as the speedy and very considerable enlargement of the spleen, which is probably the cause of a coincident, very acute leucæmic character of the blood; both of these conditions, however, disappear again during the intercurrent stage of freedom from fever. If this disease, noticed as yet only in Livonia, a district in which relapsing fever prevails, belongs truly to measles, it yet appears to me as a peculiar combination of this with relapsing fever, which thus defines the time of the appearance of the symptoms of measles and may also possess the power of modifying them. As Trojanowsky, the only observer of the derangement, does not represent this view, I mention it together with the relapses of measles.

All races of men are equally visited by measles, provided that the contagion reaches them. Thus, from Drake's communications (1854), whites, negroes, and Indians were indiscriminately attacked, after the disease had once spread throughout the southern parts of the United States, where previously only scattered cases had occurred.

Pregnancy is no defence against measles, and delivery may be hastened by this as by other diseases. Seidl even attributes to young pregnant women, toward the normal end of the pregnancy, an increased susceptibility to measles. Abortion in consequence of measles has been noticed, even with a fatal result (Rösch). Weisse observed menstruation appear for the first time in a girl of fourteen years, during the desquamative period of measles; but as there is no statement of the subsequent regular appearance of the same, the possibility is not excluded of a simple exanthematic hemorrhage from the genitals. The susceptibility to measles is said to be somewhat diminished by some chronic diseases, for instance, according to Mayr, by epilepsy, chorea, paralysis; and acute diseases appear often to exert an influence in deferring the outbreak of measles, so that the latter does not appear until convalescence from the former. Thus Weisse observed that the measles appeared, in a boy of sixteen years, immediately after erysipelas of the face, and only on those places where no exfoliation had occurred; in a typhoid patient of thirteen years, it immediately succeeded the typhus, and ran its regular course during coincident suppuration of the parotid glands; others have seen it immediately follow other acute exanthems. Acute diseases may also give rise to changes in its appearance, since it is by no means rare for measles to occur in the course of these, during even the stage of florescence of an acute exanthem, or immediately before or after this, and to mingle its symptoms with those of the coincident affection, or to exercise a modifying influence upon them. Thus the development of vaccinia is at one time uninfluenced by measles (Panum), at another decidedly protracted (van Halen, Cramer), and Fouquier observed this last even in regard to variola; whooping-cough is said to disappear entirely when symptoms of measles appear, and only to reappear after their complete expiration (Mayr). Mumps, on the other hand, according to Liverani, attacked by preference patients with measles, and increased the intensity of the disease; Mayr noticed the same in measles which attacked children immediately after cholera. Kesteven saw a girl who was suffering from typhus,[1] with from thirty to forty roseolæ, attacked during this disease by measles, which ran a typical course in the presence of the typhus. That measles can appear during the course of variola, scarlet fever, and varicella, and *vice versâ*, is shown by numerous observers, though unjustifiably denied by Hebra. Bierbaum observed measles during the course of a meningitis tuberculosa; Guersent, with a pustula maligna. Habisreutinger saw it in a boy during the period of greatest intensity of an erysipelas serpens, which began on the right foot, and attacked first the right, then the left half of the body; here it appeared partly where the erysipelas had disappeared, and partly upon the unaffected places. Barthez and Rilliet saw measles three times with erysipelas of the face, though here the former did not affect the face. Finally, the susceptibility to measles is said to be increased by many diseases, especially by affections of the organs of respiration, an assertion which is hard to prove, since the susceptibility to measles of those previously unaffected is in every case so marked.

Measles, from the universal predisposition to it among those who have not had the disease, and the subsequent almost total elimination of such a tendency after passing through an attack, is particularly a disease of childhood, and preferably of younger children. The more children there are in one house, the greater, according to Geissler, is the probability of the infection of those exposed and thus far unaffected. Most adults have already had measles as children, and therefore escape subsequent epidemics, an apparent proof that a second attack is at least improbable. Adults never affected are attacked as frequently as little children are, many cases occurring even during senility. Thus Drake (1844) saw upon a plantation several cases in aged negroes, one of them at least eighty years old; Heim, in a woman of seventy-six; Michaelsen in one of eighty-three; Schultze in an "old" lady, together with her "old" servants. The most striking evidence is afforded by the epidemic, described by Panum, of the Faröe Islands. There, namely, after the non-occurrence of measles since 1781, it was introduced from outside by a single person in 1846, and gave rise to such an epidemic that of 7,782 inhabitants only about 1,500 escaped the disease, owing to their perfect seclusion from the islands and districts infected, while over 6,000 were within a short space of time attacked. The resulting distress was of a perfectly exceptional nature, since almost every one, without regard to age, lay ill at the same time, and consequently no systematic nursing was possible. Those old people alone escaped who had been affected as children during the epidemic of 1781; while, as Panum reports, not one old person, previously unaffected, exposed to infection, escaped, although with some younger individuals this was the case. In Hagelloch, where for fourteen years there had been no epidemic, there were attacked by measles, according to Pfeilsticker, 185 out of 197 children under fourteen years of age, and of older persons only those who had escaped infection during the previous epidemic. In the light of such facts, the worthlessness is seen of all statements in regard to the susceptibility of different periods of life, where, in the preparation of statistical tables, no regard is paid to the fact of previous or non-previous infection.

---

[1] As this word is used in the German without the adjective "abdominalis" (typhoid fever) or "exanthematicus" (typhus fever), it is impossible to designate which of the two diseases is referred to, though it is probable that typhoid fever is meant. The same remark applies to all other places in the text where the simple word "typhus" is used.—TRANSLATOR'S NOTE.

With children under one year, and especially those under six months of age, measles, in spite of all denial, is decidedly less frequent, as I also can testify on the ground of my own observations. Single epidemics may vary in regard to this point, and in many of them the infection of little children occurs more frequently, yet in general the fact of the frequent immunity of sucklings is established. This admits of a twofold explanation; it may depend upon diminished susceptibility, it can, however, also be the result of less opportunity for the reception of the contagion. This last is especially the case when the suckling is the only child in the family, but by no means explains the increase of the frequency of attack in the separate stages of the first year of life. This is rather to be explained by an increase of susceptibility to measles towards the conclusion of the first year. Sucklings then possess no immunity from measles, as is stated in many reports; their susceptibility is, however, indubitably less. The following figures may serve as proof:

Le Barbillier observed an epidemic of measles in the Foundling Hospital at Bordeaux; of 33 children between one and seven years of age, 24 were attacked, while out of 40 children under one year of age only 7 were attacked. According to Bartscher, attacks of sucklings under six months of age were of very rare occurrence in Osnabrück. According to Mayr, only one fell ill in 10 among the new-born and nurslings. Bartels saw (1860) at the same time 274 patients, between one and five years of age, but only 31 under one year; nurslings often escaped altogether. Brown saw in an epidemic at Leith, among 170 cases, of which 129 were less than five years of age, only 12 patients under one year; 24, from one to two years; 49, from two to three years; 22, from three to four years; 22, from four to five years; 18, from five to six years of age. In Pfeilsticker's epidemic, except 3 totally secluded children, only 8 escaped, of whom 7 were one-half year old or less. Spiess recorded only 15 cases in the first year, and 52, 68, 62, 81, 71, 82 in each of the following years of life; Tresling, 72 in the first year, and 147, 142, 151, 139, 189, 198 in each of the later years; Kellner, 18 in the first year, and 61, 84, etc., in the following; Gummers, 11 in the first year, and 30, 33, 25, 25, 24, 22 in those following, out of 251 patients under fifteen years of age. According to Geissler there fell ill in Meerane, in 1861, out of 2,926 not previously affected children, 1,754, = 59.6 per cent.; the proportion of children under three months was 12.7 per cent.; from the third to the sixth month of age, 18.5 per cent.; from six months to one year, 35.6 per cent.; from one to two years, 56.5 per cent.; from two to three years, 61.2 per cent.; from three to four years, 67.9 per cent.; from four to five years, 70.9 per cent.; from five to six years, 72.5 per cent.; from six to seven years, 77.0 per cent; from seven to eight years, 81.3 per cent.; from eight to nine years, 78.0 per cent.; from nine to ten years, 68.0 per cent.; from ten to eleven years, 55.0 per cent.; from eleven to twelve years, 80.1 per cent.; from twelve to thirteen years, 20.8 per cent.; from thirteen to fourteen years, 63.6 per cent. (The figures of the latter years are based upon small numbers, since most children of these years had previously had the disease; the figures, consequently, are of little value, partly for this reason and partly because at this age of the children the parents might have forgotten an attack that had occurred a long time before.

Accordingly, no further doubt can exist with reference to the diminished susceptibility of nurslings, to which, according to Rilliet, Ackermann had already called attention at the end of the eighteenth century, and which near the first year of age rapidly disappears.

Individual observations, however, inform us that even the youngest children are not entirely without any predisposition. Heim reports measles in a child of four days; it was born during the severe sickness of one of the other children, began sneezing and coughing on the fourth day, and a common mild form of measles showed itself on the eighth day. Kunze saw a woman with measles, in the stage of florescence, give birth to a child, which on the fifth day of its life took the disease; both died. Oesterlen saw a remarkably mild course in patients of from eight to fourteen days; Mayr relates a case of an infant eight days old; Geissler also a mild case at the same age; Monti, one at twelve days and one at four weeks of age, in the years 1861 and 1862, one at twelve days and one at three weeks, in 1863, six cases between the ages of one and two months, between 1861 and 1865,—in all ten children under two months of age. The youngest case must therefore have been infected at birth, Heim's case even earlier; consequently children can be born possessing at once a decided susceptibility for the contagion of measles. Verson holds that he has very frequently seen measles in newborn children.

There are also single rare observations—with my best efforts I have been able to discover but six accounts of such, in which doubt does not exist as to the correctness of the diagnosis—according to which children have been born with fully developed measles, characterized by the exanthem, after the mother shortly before had been ill of the disease. Other observations are so far as this doubtful, that the exanthem was no longer visible at birth, but merely an exfoliation, which possibly was nothing more than the usual desquamation of a new-born child. In still other cases the disease of the fœtus has been presumed, simply from its uneasiness during the disease of the mother. Most writers report, it is true, the occurrence of congenital measles, but without ever having met with examples, *e.g.*, Willan, Rosenstein, Burserius, Girtanner, Reil, etc. On the other hand, S. G. Vogel has himself seen such a case, and believes them to be, as a rule, a cause of premature labor; Guersent holds the same view. Two cases of older date are related with sufficient accuracy by Hildanus and Ledelius. Clarus stated, in the Medical Society at Leipzig, that he had observed the exanthem of measles quite plainly on a fœtus, the mother of which had died during the exfoliative stage. Michaelsen relates two cases of measles in new-born children, one of which he himself observed: in this case the woman, manifesting at the time the eruption, bore a child which showed an unmistakable exanthem of measles. Seidl appears to have observed the same several times; he writes that the eruption can appear on the mother and child immediately after the birth of the latter, or the infant can be born already affected, from which it is to be assumed that the beginning of the disease dates from the final period of the pregnancy. He makes no statement, unfortunately, with regard to the number of his cases. Hedrich reports that a woman with measles was on the fourth day of the disease delivered of a girl, who was covered with spots of measles and manifested several catarrhal symptoms, sneezing, coughing, and moderately inflamed edges of the eyelids. The disease here ran a favorable course for both parties, as in the cases of Hildanus and Ledel. According to Eisenmann and others, J. Frank and Girtanner (?) have also observed cases of this kind. It would be interesting to ascertain in such cases if the "morbilli neonatorum," as might be expected, and as appears deducible in Hildanus's case from his report, have effectively destroyed the susceptibility. In regard to this I can adduce a case in which the measles of the mother, who was then five months advanced in pregnancy, did not affect the susceptibility of the fœtus; the child had a mild attack at the age of nine years, at the same time with its brothers and sisters. Höring mentions a case where a woman at term, together with four children, fell ill of measles, but forgets entirely to refer to the new-born child; presumably it remained healthy.

It was formerly often asserted that boys were more predisposed than girls; more extended observations, however, have shown that the predisposition varies, it being equal at one time in both sexes, while at other times it may be stronger in one or the other sex. Sex, therefore, does not influence the susceptibility.

That the susceptibility to measles is far more diffused than that to scarlet fever, is proved by general statistics of diseases, which everywhere show far more cases of measles than of scarlet fever. This fact is well illustrated by observations like those of Faber and Heyfelder, which showed that during a simultaneous epidemic of both exanthems, those convalescent from scarlet fever were frequently attacked by measles, while the converse was far less frequent. From this it is also evident that the tendency to the two diseases depends upon different conditions. Precisely the same has been noticed in regard to measles and rubeola (Rötheln) (Thomas and Gruel).

Single cases in all epidemics prove, moreover, that the susceptibility to measles may be absent temporarily, or perhaps even permanently. I observed during an epidemic, among about 130 cases, five children, two of whom, boys of two and three years, evinced an immunity during this epidemic, while two boys of eight and twelve years, and a girl of nine years, had evinced it as well during previous ones. Spiess also reports such cases in children of from four to seventeen years. Some few children appear, without any known cause, to be free from susceptibility for a while, but to have acquired it after a longer or shorter period. Thus Spiess alleges that such children, after having been previously exposed to the contagion with no result, fell ill, in two cases after seven weeks, once after two months, four times after two and a half, once after three, twice after four, and once after five months. That these later attacks were, however, true measles, is shown by the fact that in several cases further infection took its rise from these patients. Moore relates a triple invasion by measles of a numerous family: a son, who had passed safely through two epidemics, fell ill during the third, and infected a younger brother, who at the time of the first invasion was not born, but who had successfully resisted the second one. According to Stilling, a woman of thirty-three, whose children suffered from measles at the beginning of January, escaped at that time, but fell ill in the middle of February, at which time a daughter six years old was seized by a relapse.

Single cases of measles are partly sporadic and partly united in epidemics. A sporadic appearance occurs preferably in large cities, where the disease has become more or less endemic; yet even here at times, and then in a tolerably regular manner, it takes on the extent of an epidemic. Definite epidemics of measles appear upon low lands and in small districts removed from the great highways of commerce, and, according to the smallness and the isolated situation of these, decades and more can elapse between the separate epidemics, and in these intervals cases are absolutely lacking. For this reason, furthermore, single epidemics, when they do occur, are relatively much more considerable in small places than in larger ones, where, to a certain extent, several epidemics must be divided among those of the population who are predisposed to it. The larger a place becomes in the lapse of time, and the more considerable the commerce in it and with it, the more frequently epidemics of measles appear, and the more numerous become the intermediate sporadic cases, so that eventually in very large places the disease is always present. This condition of things is explicable by the great contagiousness of the disease, and by the universal susceptibility of individuals not as yet attacked. The number of these increases after a time by births to such an extent that it evidently often requires merely the introduction of the contagion by a single person to infect quite rapidly the larger number of those belonging to the new generation, after which, from lack of material susceptible to infection, the epidemic fades out, to appear again after years under similar conditions. In large places with much commerce the number of those susceptible can never be very considerable, since it is continually diminished by the constant introduction of the contagion from all directions, and by the more or less sporadic prevalence of the diseases thus produced.

For these reasons epidemics of measles occur with a certain periodicity in medium-sized and large places, so that in many of them now and then the outbreak of such an epidemic may be prophesied for a certain time with considerable certainty. The intervals usually vary from two to four years; the shorter the interval, the milder will the ensuing epidemic usually be; and the longer, the more intense. A mild epidemic is followed, as a rule, soon, and also out of its regular turn, by a more severe one, which compensates for the omissions of the former. We possess reports, for instance, from various places in regard to the succession of these epidemics during long periods, and from these it appears that in certain years the disease is universally prevalent, so that epidemics of measles will be found prevailing simultaneously in different cities; while, however, on the other hand, exceptions and irregularities are frequent. Sometimes the epidemics are not coincident in neighboring localities connected by constant intercourse, a proof that in this question local conditions play an important part and are frequently of determining influence. Thus we see that no universally applicable law with regard to the periodicity of epidemics of measles can be established, and the most that can be asserted is, that with the increase of intercourse and the growth of large cities they have become, in these especially, by degrees somewhat more frequent. A few examples may suffice:

According to Geissler the population of Meerane was in 1837 4,638; in 1843, 5,550; in 1850, 7,337; in 1856, 9,530; in 1861, 12,747; in 1867, 16,511; epidemics occurred in 1837, 1850, 1853, 1857, 1861, 1865, 1867, 1869; they became therefore with the increase of population correspondingly more frequent. In Leipzig measles prevailed in 1844, 1847, 1849, 1851, 1852, 1856 (1858), 1860–1, 1864, 1866–7, 1868, 1869–70, and 1872. In Dresden, according to Förster, in 1835, 1838, 1840, 1844, 1846, 1848, 1852 (1853), 1856 (1858), 1860, 1864 (1865), 1867. In Danzig, acc. to Lievin, during the years 1863–1869: 1863–64, 1865, 1868. In Königsberg, acc. to Schiefferdecker, in 1857 (1860–61), 1862–63, 1868. In Halle there were, in 1782, 24,149 inhab.; in 1852, 36,076; in 1871, 52,400; acc. to Bärensprung and Weineck, epidemics occurred here in 1784–85, 1790, 1795, 1801, 1804, 1806, 1808, 1810, 1812, 1815, 1818–19, 1823, 1828, 1831, 1833, 1836, 1838–39, 1841–42, 1843, 1845, 1848, 1850, 1852–53, 1855, 1857, 1860, 1861, 1864, 1867, 1869, 1871–72. In Zürich, acc. to Meyer-Hoffmeister: 1827, 1833, 1837, 1843, 1849. In Erlangen, acc. to Küttlinger: 1819–20, 1825, 1831–32, 1839, 1847, 1852–53, 1856. In Stuttgart, acc. to Köstlin: 1849–50, 1852–53, 1855–56, 1858, 1861, 1864–65. In Würzburg, acc. to Voit: 1846, 1849, 1854 (1855), 1860, 1863, 1866, 1868, 1871. In Frankfurt a. M., acc. to Kellner and Spiess: 1842, 1846–47, 1850, 1854–55, 1858, 1860–61, 1863–64, 1866–67. In Munich, acc. to Ranke: 1859-60, 1861–62, 1864, 1866. In Vienna, acc. to Mayr and Fleischmann: 1842, 1845–46, 1848, 1850–51, 1853, 1855, 1857, 1859, 1862, 1864, 1867, 1869. In Prague, acc. to Löschner: 1843-44, 1847–48, 1850 (1851), 1853, 1855-56, 1857–58 (1859–61 a large number of sporadic cases). The case seems to be similar in Berlin of late years: acc. to Romberg and Engel measles prevailed in 1843–44, 1844–46, 1848, 1851, 1853–55, 1857, 1859–60; acc. to Passow epidemics occurred 1862–63, 1864–65, 1866–67; in Formey's book, on the other hand, the periods are not of two but of three years: 1786, 1789–90, 1793. In Geneva, acc. to Rilliet: 1832, 1838, 1842, 1846–47. In London, on the other hand, the annual mortality from measles was from 1856–1866 between 17.54 and 36.96 per thousand; it prevailed therefore with slight variations continuously. In contrast with these short intervals, we may again refer to that of Hagelloch (fourteen years) and that of the Faröe Islands (sixty-five years); on the Cape of Good Hope, also, measles reappeared, after a pause of thirty years, in consequence of a fresh introduction. In Iceland epidemics occurred only in 1644, 1694, 1846; at Madeira they were unknown until 1808.

Like all acute infectious diseases, and especially variola, measles may also appear at times with a general, almost pandemic spread, to then disappear again more or less completely for a longer or shorter time. According to Hirsch, such universal outbreaks have been observed already several times during the current century; so for instance in 1796–1801 in a large part of France and England, in 1823–24 in Germany, in 1826–28 in the Netherlands and Germany, in 1834–36 and again in 1842–43 in the larger part of northern and middle Europe, and finally in 1846–47 in almost universal extension over northern and western Europe and in North America.

Apart from their frequency and their tolerably regular succession, the form also of single epidemics of measles is extraordinarily characteristic. They have mostly a short duration, and increase, when once under way, very rapidly to quite a marked extent, at which acme they tarry with slight variations for but a short time, then just as speedily diminish, and either vanish entirely, or, as is the case in large cities, make room for more or less numerous sporadic cases. These abrupt increases are, by the way, not exceptional, but present in all great epidemics; in smaller epidemics, resembling rather numerous sporadic cases, the type is more or less irregular, with several maxima, and the duration of the epidemic is then, as a rule, also more protracted.

Epidemics of measles can arise at any season of the year, yet statistical tables show that they occur more frequently during the cold season.

According to Hirsch, of 309 epidemics of moderate extent, 96 began in winter (28 in December, 54 in January, 14 in February), 94 in spring (43 in March, 28 in April, 23 in May), 43 in summer (19 in June, 16 in July, 8 in August), 76 in the autumn (16 in September, 34 in October, 26 in November).

Although conditions of temperature, which favor the occurrence of catarrhal forms of disease, would seem from this to exert an essential influence upon the rise and spread of measles, yet a series of facts goes to show that this disease can also arise entirely independently of the cause cited, and that the spread of an epidemic once started certainly ensues at any time and under all meteorological conditions. Perhaps the better ventilation of sick-chambers during mild weather, and the consequent diminished concentration of the contagion, with the apparently slight vitality of the same, may be the reason why the introduction of the poison often produces no effect; while the inferior hygienic conditions pertaining to the cold season may be far more favorable to the development and spread of an epidemic of measles. It is certainly a fact, that in thickly peopled and badly ventilated localities, by far fewer individuals with moderate susceptibility (such as nurslings, and a few older children) escape than under the opposite conditions.

The seasons seem to exert, however, some influence upon the course of epidemics of measles in respect to their character, their mildness or malignancy, and the mortality from them. This must not be understood as meaning that the weather affects the mildness or severity of the contagion, which is not the case. The influence exerted is rather upon the occurrence of complications and sequelæ, and the nature of the convalescence. The cold season, and still more the changeable weather of spring and autumn, decidedly favor the production of, and interfere with the recovery from, affections of the lungs, which may also possibly be influenced unfavorably by the existence of a catarrh of the air-passages acquired before the attack of measles; while the hot season, though to a much less degree, occasions the development of catarrh of the intestines, thus modifying the normal course of the measles. There are no definite statistics with regard to this, partly because they are in most instances based upon old cases, while in this important question the more rational mode of treatment of modern times exercises a decided influence, and must consequently be taken into consideration.

The question of the time at which measles are most infectious and the contagion most widely diffused, stands in intimate relation with the question of the duration of the stage of incubation in those who have been infected.

Since the precursory stage of measles begins, as a rule, with marked fever, it is generally easy to determine the point at which the stage of incubation terminates; for by incubation is understood that period only which elapses between the infection and the evident commencement of the symptoms of the disease, that is, of the fever. The entire prodromal stage was formerly often comprehended under this name, and the period of incubation was thus understood to extend up to the eruption of the exanthem, a view which has occasioned much confusion.

It is quite difficult, usually impossible, to determine in a single case the moment or even the day of infection. It takes place, as a rule, in the family circle or at school, that is, under conditions which involve frequent or continuous contact with the contagion. Single observations, however, exist in which a single exposure, or that of only a single day is reported, and which therefore permit us to regard a fixed time as that of the infection. The persons who in these cases occasioned the infection were partly in the precursory, partly in the eruptive stage; at some time, therefore, during these two stages, the infecting material must have been effectively generated and thrown out. If, however, the fact is regarded that the contagion may adhere to material objects, those for example of the sick-room, and preserve its activity for a time as yet undetermined, the possibility certainly cannot be excluded that a material thrown off during the prodromal stage may occasion infection some days later during the stage of eruption, at a time even when it is conceivable that the elimination of contagion from the diseased organism no longer takes place. The symptoms on the part of the mucous membranes are, however, similar in the prodromal and eruptive stages, with the addition merely of the exanthem in the latter stage, and there is no ground for doubting the possibility of their giving rise to an effective contagion, but in no case as yet has proof been furnished that that substance, which during the eruptive stage occasioned the infection, had been just before eliminated and worked contagiously therefore in a perfectly fresh condition. Such proof could only be furnished if a child, at the commencement or height of the eruption, should be washed, invested with clean and not-to-be-suspected clothes, transported, without the accompaniment of any infected object, to a healthy locality, and there produce infection. Since the disease in those infected begins only at the expiration of a stage of incubation, consequently at a time when the infecting patients are convalescent and their skin exfoliating, it was formerly held that the infection occurred especially or solely during this period, and not at any earlier one. That this opinion is completely erroneous is shown by those numerous observations in which contact has occurred only during the first period of the disease, the exposed person, however, becoming infected; this opinion, therefore, is at present given up entirely.

On the other hand, it is clearly proved that effective contagion is produced at the beginning of and during the prodromal stage, and it is precisely at this period that the greatest spread of the contagion takes place. Evidence of this is afforded by the only slightly varying duration of the incubative stage ascertained in the few cases mentioned above, where the contact of the infected person with the source of contagion was but for a moment or for one day. If we reckon, in common cases where a family is infected, fourteen days back from the outbreak of the exanthem in the second child attacked, we come noticeably often upon the first or second day of the prodromal stage, or the last day of the incubative stage in the original case. It is especially desirable, and would not prove a thankless task, for country physicians in secluded districts to increase the small number of standard observations by further cases accurately reported after careful investigation.

The majority of these few cases have been reported by Panum, who made his observations upon the Faröe Islands under, it must be confessed, the most extraordinarily favorable circumstances. In all cases thirteen or fourteen days elapsed from the day of infection to the commencement of the eruption, and that too whether the infection had taken place during the prodromal or the eruptive stage, upon which latter circumstance Panum lays needless stress. The cases in question concerned only: the ten men from Tjornevig (exanthem at end of fourteen days), a woman in Welberstad (fourteen days), the two young men from Hatterwig (thirteen days), the first young man from Selleträd (fourteen days), his brother and other people in the village (thirteen days), nine people in Fuglefjord (fourteen days), the crew from Dimon (fourteen days), the two men from Skaalevig who fetched the physician (fourteen and thirteen days). As compared with these, in all nearly forty cases, the remaining literature furnishes but a scant return, since, for the most part, a contact of several days between the person infected and the first patient had taken place, and the exact estimation, to a day, of the duration of the incubative stage is therefore impossible. I have been able to find only very few serviceable reports; among these are: the first case by Gregory (fourteen days), a child by Mayr (fourteen days), two children by Dumas (from ten to twelve days, according to Simon's report), a girl of eighteen years by Küttner (ten days to the time of the "eruptive fever"), and two boys by Spiess (fourteen days). In addition to these cases many others exist where, on the first outbreak of measles in families or hospitals, children were at once removed, but fell ill nevertheless and manifested the exanthem about fourteen days later; cases which, however, do not preclude the possibility that the stage of incubation may have been abnormally protracted, inasmuch as the infection may have taken place in the prodromal stage before the outbreak of the measles.

If we bear in mind the fact that the duration of the prodromal stage of measles lasts three or four days, the stage of incubation, as shown by Panum's observations, must be considered as extending over ten days. This period represents the normal duration of the same. With this corresponds very well the experience, unaccompanied by special instances, of Abelin, who noticed that when a case of measles was brought into the hospital the disease began among the other children in nine days.

Others have endeavored to establish the duration of the incubation of measles in this way: in families, where a child had introduced the disease, they noted the number of days from that of the illness or eruption of the first child to that of the same in the others. It is evident that this manner of reckoning does not afford any trustworthy estimation of the duration of the incubative stage, since the beginning of the illness and the eruption are not always equally widely separated as to time, and the infection of the subsequent cases can clearly have ensued at very varying periods, namely: 1, *before* the illness of the first child, from the same or another source, and particularly from the contagion in the clothes of the first child; 2, *during* the course of the disease of the first child, from the infectious material produced by it, and this either at any time of the prodromal or of the eruptive stage; 3, *after* the disease of the first child,—or at least at a time when the child no longer diffused any infectious material,—from contagion which it had produced during its illness and which was communicated to the second child by means of inanimate objects or otherwise at second hand. Since, however, it may be taken for granted, from the great contagiousness of measles even during the prodromal stage, and from the great susceptibility to measles in those never attacked, that the infection will occur as soon as possible, we may expect, in such estimations as the above, to meet no very varying numbers, and in the majority of cases to observe nearly the normal duration of the incubative period.

In this connection I note the following results: Pfeilsticker, proceeding on the assumption that the infection of those subsequently attacked took place on the first day of the prodromal stage, found an interval of from thirteen to fifteen days between the infection and the exanthem; Girard, with the exception of three cases where it lasted sixteen days, one of thirteen or fourteen days. In the six cases of Harnier, where the infection could only have occurred during the prodromal stage, the intervals between the eruptions were from eleven to thirteen days. In a carefully controlled case by Rilliet, the interval between the first signs in two children was twelve, that between the eruptions ten days, and the exanthem of the second child appeared fifteen days after the commencement of the prodromal stage in the first. Spiess, on the other hand, observed only the time of the outbreak of the two exanthems, and found the interval to be, in one hundred and forty-seven cases, one hundred and seventeen times between ten and fourteen days, eight times in nine days, and twenty-two times between fifteen and eighteen days; Salzmann found, reckoning in the same way, in twenty-five cases infected from a single source, three times in nine, eight times in ten, thirteen times in eleven, and once in twelve days; according to Kerschensteiner, the exanthem of the second series in thirty-seven families appeared thirty-four times between the tenth and twelfth days after the outbreak of the first eruption, and once each on the eighth, fourteenth, and fifteenth days. I frequently observed in the two series the beginnings of the prodromal stages and the maxima of the exanthems, and found for the first an interval of ten, for the latter one of nine or ten days; these numbers, therefore, coincide exactly,—or at least this is true of the majority of them,—with the more accurate duration of the incubation given by Panum.

Observations even so ambiguous as these may serve to confirm the rule that the incubative stage of measles possesses a definite duration of about ten days; it is not allowable to infer from them the opposite.

An interval between the maxima of the exanthems of the infecting and affected children, which far exceeds the normal standard, may be explained by the at first slight and subsequently increasing susceptibility of the second child, so that it becomes infected by the contagion which still adheres to objects; it might, however, also be explained by an unusually long duration of the incubation. Against this last is the fact that such a one was never yet confirmed by undoubted observations;—a further reason for laboring for the greatest possible augmentation of their number. Furthermore, it is to be remembered that Panum's figures are derived from measles in adults, not in children.

How certain unusual observations are to be explained must for the present be left undecided; it is probable that the contagion adhered to objects which did not at once come into contact with the person subsequently affected. Thus Roux observed an epidemic of measles, which broke out among the healthy occupants of a vessel seventeen days after leaving the harbor; Tuffnell, the isolated affection of a soldier who for forty-five days had been in prison. In both cases the diagnosis is said to be indisputable. Possibly existing chronic diseases sometimes influence the extent of the incubative stage: thus, according to Mayr, the rickets. Emmert reports a case where a boy of ten years, just recovered from an acute rheumatism, fell very ill after an incubation of two and a half weeks.

PATHOLOGY.

Anatomical Changes.

Measles are characterized by an eruption upon the skin of red spots, accompanied by catarrh of the mucous membrane of the upper air-passages and by a brisk fever.

In normal cases perfectly developed measle-spots have a diameter varying from one-twentieth to two-fifths of an inch; their form is roundish, long, half circular, or like a half moon. Their margins are rarely rounded off, generally variously indented, and frequently even provided with offshoots, not fading off into surrounding parts, but sharply defined. Their color is more or less rosy red, sometimes with a light shade of blue, at times dark red. They are usually discrete and separated by healthy, pale tracts of skin; but where closely contiguous their margins can in various ways become confluent, and thus produce at one time a more or less uniform redness over a large extent of skin, on which can still be recognized, however, the original spotted character by clearer and darker streaks, as well as by single pale points remaining free; at another, a marbled injection of the skin in irregular configurations, separated by numerous pale streaks and spots. At times, in confluent measles, moderate turgescence of the skin takes place. In no case does the redness occupy the entire superficial area of the body uninterruptedly, only single tracts are at most confluent, the others show the usual spots. A certain resemblance to scarlet fever can at all events be occasioned in severe cases by an extended and almost universal confluence. The single spots are very slightly raised, and very often have in the centre of each a miliary papule, which frequently occupies the place of a hair, and rises almost imperceptibly above the spot. The spot disappears on pressure, but when this is removed returns immediately with the same form and similar characteristics. Many spots contain several papules. With an intense exanthem these fine papules are also more evident, but rarely so strongly developed as to mislead a person into confusing them with forming variola papules. They characterize to a marked degree the eruption of measles, and distinguish a confluent exanthem resembling scarlet fever from a true case of the latter. As a rule, the hyperæmia around the papule is somewhat darker than at the margin of the spot itself.

Mayr and Hebra seek for the anatomical basis of the measles papule in an inflammation of the sebaceous follicle of the skin, which excites a superficial capillary injection of the contiguous parts, which is generally limited by the furrows of the skin, thus losing the form of a circular inflammatory areola. The hairs which are present in the inflammatory tract are merely incidentally so; if the hair-follicles specially participated in this inflammatory process, the scalp would necessarily be the proper focus of the exanthem, and the palms of the hands and the soles of the feet would remain free, neither of which is the case. This opinion, not based otherwise upon anatomical investigations, is not shared by G. Simon. He examined a papule cut, together with a small portion of the subjacent cutis, from a boy with measles. It appeared that the epidermis was unaltered, and not separated from the cutis; this latter bulged somewhat at the site of the papule; why, was not ascertained. Abnormal elements were not present, with the exception of very small, roundish molecules, unaffected by acetic acid, between the cutis fibres; these themselves showed no change, the papillæ were not enlarged, and it was presumed therefore that the papule resulted merely from a collection of fluid. On the upper portion of the hair sac no change was found, nor any swelling of the sebaceous glands. Neighboring papules seemed often to unite in a group, which was then, as a rule, surrounded by a more extended hyperæmia than where single papules only had been developed. The exanthem acquires a somewhat abnormal appearance when the papules are not grouped but discrete, and the roseola of measles is formed around each separate papule. This is especially the case in young children and nurslings. When the roseolæ are here so large as to be almost confluent, the appearance of the eruption is rather suggestive of scarlet fever. On the other hand, an unusually marked development of the papules often occurs in anæmic, scrofulous children, and in such cases very considerable, entirely or nearly pale, papules frequently appear near and between the usual hyperæmic ones. The sight of the hyperæmia appears to be in the deeper strata of the rete Malpighii.

The spots of measles are developed upon all parts of the body, on the face and trunk more than upon the extremities, especially the lower, where in light cases they are very scattered, and somewhat smaller. The confluence of separate spots is usually confined to the face, where also, as the most vascular part of the skin, they are usually most developed; and their redness, which is tolerably uniform throughout other parts of the body, is here often somewhat brighter. They have no regular arrangement, and are scattered about, the smaller and larger ones alternating in various ways. It is rare that other parts are more affected than the face. Now and then no difference can be perceived between this and the whole or single parts of the trunk, and in this case there may be a more or less universal confluence, as in scarlet fever, even at times upon the scalp also. This occurs only in very severe cases. The eruption upon the gluteal region is often of a darker color, and its appearance modified by an erythema arising in consequence of pressure. The buccal region, palms, and soles are covered with spots like the rest of the body, the genitals of boys and girls to a less extent. The exanthem varies but little as to quality and quantity upon the flexor and extensor aspects of the extremities; any existing difference is accidental. I have often seen in anæmic patients, with a delicate skin, an eruption which was in all parts of the body of a light shade of rose color, the extent and swelling of the spots, nevertheless, leaving nothing to be desired.

If the exanthem is specially scanty, the single spots are never equally diffused over the whole body, but grouped upon a smaller or larger surface, generally discrete, sometimes slightly confluent. Neighboring surfaces of equal extent may be free or slightly spotted.

The diversity of form in individual cases depends upon various deviations of the same in regard to the size, height, and grouping of the papules, to the extent and intensity of the consecutive hyperæmia, to the swelling of the hyperæmic spots, to the manner of mutual position on the part of the spots, and can also be due to variations in the spread of the exanthem upon single parts of the body, or to the occurrence of bruises and the development of vesicles upon the site of single spots. Mayr and Hebra distinguish: *Morbilli læves*, smooth, simple measles, without especial elevation of the follicle, all the spots clearly isolated; *M. papulosi*, with a more evident papular formation, so that this feature specially defines the character of the eruption; *M. vesiculosi seu miliares*, when the eliminating ducts of the follicles, filled by fluid exudation, protrude in the form of fine, transparent miliary vesicles, giving to the eruption an appearance resembling miliaria; this is especially the case when perspiration is profuse; *M. confluentes*, due, according to these authors, to the outbreak of such a number of spots or papules that the free interspaces are reduced to a minimum; *M. hæmorrhagici*, where the efflorescences represent spots or papules of a dark red color, not disappearing on pressure; these result from capillary hemorrhages.

The outbreak of the exanthem does not always take place at a similar period of the fever, while the point of its highest development, the maximum of the exanthem, is a nearly fixed one. Thus, the earliest signs of the eruption, at first in a thoroughly undeveloped condition, appear not infrequently upon the first day of the febrile period, more often on the second or third; in the larger half of the cases, however, they appear late, from one-half to one and a half days before its maximum. As the point of origin of this single manifestation, I recognized in most cases a faintly hyperæmic swelling around the orifice of a sebaceous gland, the papular character of the exanthem thus being pronounced at the outset. A faint hyperæmia, without swelling, existed only rarely at first, more often a pale papule without hyperæmia. Variations of this kind appeared usually, not isolated upon single parts of the body or extended areas, but near to and between the hyperæmic papules. When the development of the exanthem is rapid, these abnormal appearances speedily develop into the usual spots of measles, and yet I have also seen in several cases the disappearance, during convalescence, of a considerable number of papules which had remained pale. On the other hand, the spots, even in the lightest cases, never remain in their original purely hyperæmic condition.

The papules show themselves first of all upon the face, especially on the chin, cheeks, forehead, and temples, furthermore over the processus mastoideus, and very commonly also upon the scalp, at times upon the occipital region. Somewhat later, but still at an early period, they also often appear upon the throat, neck, and the upper parts of the breast and back; only very rarely, and then faintly, they appear upon the last-named or other parts at first.

When it appears quite early the exanthem at first changes but slowly, and extends just as slowly to other parts of the body Thus it may develop and remain for days in a moderately developed condition upon the face alone. A quicker increase then takes place at or soon after the beginning of the marked increase of temperature which characterizes the acme of the disease. While previously a slight hyperæmia had begun to form very slowly around the swollen follicular orifices, it now spreads very rapidly; the red areolæ around contiguous papules run together, and swelling of the hyperæmic spots occurs over regions of varied extent. The previously unaffected parts are now attacked in rapid succession. While roseolæ appear upon the face around the original papules, there arises on the trunk, and soon also upon the extremities, a formation of fresh papules, the further development of which progresses speedily. In this way it is very common for the maximum of the exanthem in the face to exist in a state of perfection at a time when its most marked development has been also reached upon the other parts of the body.

When, however, the eruption first appears at a late period, namely, during the acme, its extension over the body is more rapid. In a premature outbreak the development and extension of the single spots can be followed step by step. Where this is delayed, there appears at once, and almost over the whole body at the same time, an exanthem, developing speedily, especially in regard to the formation of roseolæ, where half a day before only traces could be observed, or not even these. Its increase in intensity and extension is rapid, and its maximum is soon attained.

The duration of this maximum, that is, of the greatest development of the eruption on all parts of the body at once, is about half a day, more or less. It is usually observed only at evening, and is then of shorter duration, since by the next morning retrograde metamorphosis of the exanthem has, as a rule, already begun; while, if it appears in the morning hours, it usually, under the influence of the evening increase of fever, remains until night, or even perhaps increases somewhat at evening. I have never observed the duration for twenty-four hours of a true maximal development. I find, furthermore, that others also describe the duration of the maximum as being very rarely longer than this.

The retrocession of the exanthem takes place usually somewhat disproportionately on the different parts of the body. In general it is the rule that the parts first attacked are also the first to become normal again. Twenty-four hours after the ebb of the maximum of the exanthem, we find usually that the head and trunk, also the upper halves of the extremities, are pale, while hyperæmic spots still remain upon only the lower legs and feet, forearms and hands. As a rule, however, the redness of these parts will be found also to have diminished long before. If the maximum is rather protracted, the spots upon the extremities are often still slowly progressing, while the face has already begun to fade out; one sees, then, only a scanty pale red exanthem on the face, while on the lower part of the body the spots are larger, redder, more elevated than a few hours before. Such slight deviations take place especially when the eruption of measles is intense. If during the decline of the fever a considerable exacerbation should abnormally recur, one often sees the faded redness return upon those parts of the body recently and severely attacked; and that, too, to a marked degree, even if not perfectly in its former strength. This is likely to be the case on the trunk, and on the upper parts of the arms and legs, especially the hinder parts of these which are kept pressed and warm by lying in bed. This new and less marked redness always lasts but a short time; the influence of the increased activity of the heart's action produced by the fever is soon overcome by the tendency to contraction of the vessels of the spots of measles, and the retrocession of the exanthem again proceeds. When the duration of the defervescence lasts for a day and a half, the usual inconsiderable exacerbation exerts either very little influence or none at all, upon the color of the exanthem, nor does one notice, as a rule, any striking return of the redness upon the occurrence of feverish complications at a still later time; the eruption rarely, under such circumstances, appears with any degree of distinctness, or at most the spots are faded and not clearly defined. A rapidly progressing fading of the exanthem, after the ebb of its short maximum, may be considered as the rule, and a renewed redness of spots which had faded, as the exception.

I have never had an opportunity to convince myself of the connection of a speedy fading of the spots with the sudden occurrence of a complication. A simple, rapidly progressing paleness of these can certainly not be considered anomalous.

When the exanthem is not excessively crowded together, and has become confluent only at the height of the maximum, from the great spread of the roseolæ, this confluence usually disappears during the fading stage, and the centres of the spots, which before were the most deeply red, alone retain any color. The very slight swelling of the spots, and that especially of the nodular protuberances, disappears simultaneously with their loss of color.

It is only in rare cases, and where the hyperæmia is very slight and of short duration, that the roseolæ disappear without leaving some traces; they usually leave behind a yellowish or brownish stain, signs of which are also often quite evident upon pressure, even at the time of the maximum of the exanthem. These stains last—according to the intensity of their color, occasioned by the presence of red blood-corpuscles, or at least of the coloring matter of the blood—for a varying number of days; and from them, sometimes even after from eight to fourteen days, the previous existence of the exanthem may be determined. Their color becomes less by degrees, and vanishes imperceptibly,—to a certain extent, undoubtedly, with the epidermal layers, which at the time of the exanthem were still deeply situated. The pigmentation is generally deeper the redder the previous exanthem was; and from it alone is the latter still recognizable upon the dead body.

More noteworthy are the colorations produced by actual extravasation of blood on the site of the spots that frequently appear during the retrograding stage of an attack of measles, which otherwise runs a perfectly normal course. Such hemorrhagic stains can appear upon individual parts, or upon the whole body, and then more or less upon all or the majority of the places previously simply hyperæmic. Their color varies between wine-red and clear to dark violet, in the most various shades, and is not, as a rule, of the same depth upon different parts of the body. They appear to be composed, at one time, chiefly of single, larger or smaller points of blood; at another, they represent more uniformly colored areas, with well-defined margins, as in the previously existing spots; if these were confluent, the same can be the case here. The hemorrhages frequently appear even during the maximum of the exanthem, so that this shows at that time an unusually dark tint, and does not disappear entirely on pressure; the hemorrhagic nature of the spots becomes perfectly evident only upon the fading of the roseolæ, and it can, then, in spite of the disappearance of the hyperæmia of their sites, be still visible, just as in the florescent stage of the eruption. Through the customary transformation of the coloring matter of the blood, they assume by degrees a paler bluish, and dirty yellowish tinge, and disappear like common measles, more or less quickly, according to the depth of their hue. Extravasations of blood depend doubtless upon the fact that under the influence of the contagion of measles the walls of the vessels become temporarily, and usually only locally, more fragile, or permit at least a more profuse escape of red blood-cells; any permanent injury from this I have never seen, nor have Veit, Hochmuth, and other observers. A dissolution of the blood, as the cause of these benignant hemorrhagic effusions, is at all events entirely out of the question.

When the exanthem is strongly developed, a little vesicle appears now and then upon the summit of the papule, especially upon the trunk and under the influence of profuse perspiration. Heidenreich states that the contents of these miliary vesicles of measles have an acid reaction, but this has not been found by others to be the case. The inoculability of these contents has been asserted on several sides. I have never seen in any patient the entire eruption marked by a vesicular conformation.

Exfoliation of the epidermis takes place generally only to a moderate extent on the face, but here tolerably regularly, especially on the forehead, cheeks, and nose; a by no means inconsiderable exfoliation occurs, however, at times even upon the whole body. Profuse perspiration, frequent cleaning of the body, and baths generally interfere with the occurrence of a perceptible desquamation, while the nature of the eruption has a less influence upon its development; yet, as a rule, an intense exanthem and marked desquamation coincide; exceptionally, with an equally severe exanthem there may be no exfoliation, or with a mild exanthem, on the other hand, some exfoliation may take place. The exfoliation, according to my experience, takes place only upon the sites of the spots, and is, therefore, always branny or in fine scales; larger scales occur only with confluent exanthems, but yet never to the same degree as in scarlet fever. In measles the skin does not peel off in large lamellæ from the fingers, palms, toes, and soles. Desquamation may be apparent very early, a few days after the maximum of the exanthem, while the redness of the spots still exists; at another time it only appears after complete fading. As a rule, it lasts some days, rarely protracting itself into the second week, or disappearing as early as in one or two days. Thus its appearance and duration are both undetermined. According to my experience with scarlet fever, I hold it as probable that, in the oft-cited cases or epidemics with quite anomalous symptoms or groups of symptoms, the coincidently observed desquamation in large lamellæ and of long duration is to be explained by the confounding of measles with scarlet fever, or by the combination of the two diseases.

As a rule, *the mucous membranes of the nose, the throat, the upper air-passages, and the conjunctiva* are attacked, in measles, and often much sooner than the outer skin. The catarrh of the upper air-passages in particular is, in all parts of the earth where measles has as yet been observed, a so constant manifestation, that it justly merits the attribute of a pathognomonic symptom, a fact especially to be considered in the diagnosis of measles in the colored races. How far the hyperæmia of the mucous membranes in measles can extend in normal cases towards the lungs and the œsophagus, and in what form it exists upon more remote and less accessible mucous surfaces, is unknown; on the visible mucous membranes the following condition may be seen:—

Sometimes, at the end of the stage of incubation, and certainly in the beginning of the prodromal stage, or soon after, there is found, corresponding to the symptoms which appear at this time (sneezing, coughing, and running from eyes), a congested condition of the mucous membrane of the eyes, nose, throat, and larynx; this steadily increases, and becomes most intense just before the outbreak of the exanthem, and in the beginning of the period of eruption. During the maximum of the exanthem, or, where the outbreak is intense, at the latest with the close of this maximum, this hyperæmia begins to fade out, generally disappears rapidly, and is entirely gone at the end of a few days. Earlier or later,—frequently by the second or even by the end of the first day of the disease, but rarely as late as on the third day,—this hyperæmia has commonly extended from the back nasal passages to the neighboring portions of the soft and hard palates and the buccal cavity generally. When the *palatal mucous membrane* is first attacked, there may often be seen, together with single, somewhat dilated vascular twigs, a larger or smaller number of very minute hyperæmic points, which spread rapidly. This process affords a peculiar appearance during its highest development at the beginning of or just before the eruption. The mucous membrane is reddened, especially toward the uvula and the palatal arch, and upon it may be found, to a greater or less extent, dark red spots from the size of a pin's head to that of a bean, or larger, of very irregular contour, varying in distance from each other, and with faded margins, which, for the most part, are connected at single points with their neighbors by faintly colored bands; a nearly universal confluence is more rare. At the close of this process, which lasts but a few days, several more marked swellings, red, and of the size of a millet seed, frequently arise, especially upon the darker and more uniformly reddened back portions, just as they also occur, in varying numbers, and irregularly scattered, in an ordinary chronic catarrh. These papules have no close relation with the faintly outlined red spots of the mucous membrane. This spotted condition, owing to the deeply injected condition of the mucous membrane far back, is here very indistinct; it is more apparent where the injection is less, and especially in the parts further forward; there exists, however, no special resemblance between this condition of the mucous membrane and the typical form of the eruption upon the skin. The retrocession of this redness of the mucous membrane occurs often simultaneously with, but usually earlier than, that of the exanthem; it may be influenced, however, by the intercurrent, abnormal development, during the process of the measles, of some further more intense derangement of the throat, but only by such a one. With the usual normal course of the affection, we see at one time only a few injected vascular trunks, more often some punctiform extravasations in addition, which quickly disappear and seem more numerous, though also much smaller, than those hemorrhages upon the skin, which are merely faint pigment stains. On the *buccal mucous membrane* we notice sometimes a faint injection of single places, of the lips by preference, elsewhere often nothing at all abnormal, while at other times an exanthematous, blotchy formation may be seen, but much less distinct than on the palate. The same condition obtains with the *conjunctiva;* a slight general injection is never absent, but more distinct spots I have never been able to make out; the ciliary margin and lachrymal caruncle are usually considerably reddened. When the eruption upon the face is intense, with high fever, the redness of the conjunctiva is also usually considerable, and occasions a slight or more marked œdema of the lids. Nor could I detect anywhere what might be called a blotchy character of the mucous membrane of the nose and throat, the redness of which fades somewhat more slowly than that of the palate. Observations have also been made upon the laryngeal mucous membrane of living persons: Rehn found at the beginning of the eruption a tolerably uniformly diffused, certainly not blotchy, redness; Stoffella (presumably at different stages), an equally diffused intense redness; Gerhardt, on the other hand, noticed in several cases in the prodromal stage, simultaneously with the blotchy redness of the palate, a similar condition also of the epiglottis and larynx. In dead bodies a blotchy redness was found several times by Steiner upon the larynx and bronchi, by Wilson, Eisenmann and Rayer on the trachea and bronchi, by Gerhardt on the hinder wall of the trachea. I cannot admit, from these contradictory and in part insufficiently characteristic results, as well as from the somewhat imperfect statements, any similarity of this affection of the mucous membrane with that of the external skin, and, consequently, do not recognize the perfect justness of the term "exanthem of the mucous membrane;" but I willingly concede that, since the peculiarities of the process upon the mucous membrane are unmistakable, one does not need to be so precise in one's application of the conception of the exanthem of measles here as upon the outer skin, since not its form, but its mere appearance at all, is of any significance in regard to a diagnosis. Possibly we have to do with a mixed process: together with an undoubted simple catarrhal condition of the nose, throat, and upper air-passages, there may exist, supposably, an indistinct, blotchy, exanthematous one, especially upon the palate, the evidence of which in the portions further back is often more or less obscured by the intensity of the catarrh.

The *tongue* offers in contrast with scarlet fever nothing typical; it is usually furred, and its turgescent papillæ often stand out somewhat more prominently.

Of the older authorities, Heyfelder in particular mentions an eruption like measles upon the mucous membrane of the duodenum and jejunum, sometimes also, at the same time, of the stomach and ileum; Weber, Eisenmann, Fuchs, and Lieutaud also mention it. In modern times Steiner speaks of a blotchy redness of the intestinal mucous membrane in children dying during the stage of florescence, and compares it to the exanthem of the skin, an opinion for and against which an equal number of grounds may, according to Volz, be adduced. It is not yet established whether such affections, perhaps of a lighter nature, can be found also in normal measles; for this reason they may in the meantime properly be recorded here. Fuchs says that at times even the genital mucous membrane is covered with numerous red, somewhat puffy spots overspread with mucus. Henoch and Chomel also relate such cases. These statements in regard to an affection of the mucous membrane, visible even on the dead body, should attract our attention all the more from the fact that, except where the measles have been hemorrhagic in character, almost no other lesion beyond the exanthem is noticed after death. Many authors assert that a general furfuraceous desquamation of the epithelium of the buccal cavity and of the kidneys is a normal appearance in measles, although they admit that it is present here to a much less degree than in scarlet fever. The affection of the mucous membrane is more often erythematous than follicular; in intense cases, however, the latter may exist, as is shown by some reports of autopsies. More severe affections of the mucous membrane belong to the decidedly anomalous forms of the measles process.

Stiebel mentions a blotchy redness of the *pleura pulmonalis.* This was covered on both sides, before as well as behind, with round, red spots, partly discrete and partly arranged in groups, but never blending; they were situated immediately beneath the pleura, their contours were sharp, and they were not altered by inflation of the lungs. They resembled an extravasation, and were partly surrounded by small ramifications of vessels; but were, however, plainly to be distinguished from extravasations. He observed them to a similar extent in four autopsies.

The *spleen* in measles is found to be moderately swollen; in the same way the lymphatic glands appear frequently to become somewhat enlarged even before the exanthem.

The *blood* is thin and fluid, dark, poor in fibrine, and shows in fatal cases a great tendency to infiltrate the tissues. The number of the red blood-corpuscles is diminished, that of the white at times sensibly increased.

SYMPTOMATOLOGY.

The course of measles, as of every other infectious disease, possesses a definite type, which is most clearly marked in uncomplicated cases of medium severity. Normal cases are such as follow the ideal type in all essential points, and therefore naturally constitute the starting-point of the description.

There are two factors, without the normal condition of which an attack of measles cannot be considered normal: the *exanthem* and the *general disturbance*, which is portrayed in the manifestations of the fever. Inconsiderable derangements of the course of the disease do not affect both, and should the normal type be lost through some extraordinary influence, it is the more sensitive fever which is first or perhaps even alone changed.

Normally the stage of incubation is that of perfect latency of the disease, consequently without fever and free from local symptoms. It may be regarded as a slight and unimportant anomaly, when now and then at its conclusion, or at another time, an inconsiderable rise of temperature occurs with or without moderate symptoms of catarrh of the air-passages. The latter is not infrequent, especially at the close of this stage, and it is conceivable that, even at this time, infection may be occasioned by means of the secretion. Somewhat more considerable elevations of temperature may also at times occur under the form of an ephemeral fever, with various slight feverish symptoms; but its short duration prevents any effect upon the further course of the disease. These febrile manifestations may perhaps arise from incidental causes in consequence of an increased susceptibility on the part of the individual infected.

The febrile period in measles may be divided into two principal stages, according to the absence or presence of the exanthem: the *prodromal* and the exanthematic or *eruptive stages*, from which also a stage of florescence is sometimes distinguished.

The duration of the *prodromal stage* is, in normal cases, three, at most, four or five days. During its continuance, in many cases, no evidence of the typical exanthem of measles is yet visible; in others, indications at least of this appear on different parts of the body, principally upon the face; this happens especially on the third day, and sometimes even on the second, but rarely on the first. One notices here, for instance, a larger or smaller number of the minute nodules mentioned above; these may increase slightly toward the end of the prodromal stage, but the typical blotches never form around them thus early. The nodules are generally so small as to escape observation, or call for the most minute examination. This is less the case with the blotchy affection of the mucous membrane, which has already been described. This usually appears before the exanthem, and is even frequently well developed on the third, or possibly even on the second day of the prodromal stage; it more rarely appears simultaneously with the spots upon the skin, on the fourth or fifth day of the disease. On the first day it is more common to find some slight evidence of the development of this affection of the mucous membrane than to find indications of the subsequent exanthem. On the other hand, the anatomical changes which are the cause of the typical prodromal symptoms—the catarrh of the mucous membranes—is usually present from the first. These changes of the mucous membrane and their sequelæ are very rarely entirely absent in normal measles; they take place, at the earliest, at the commencement of the prodromal stage.

The prodromal stage begins suddenly, as a rule, during perfect health, or after previous slight catarrhal disturbances, with fever, cough, snuffles, sneezing, photophobia and pressure in the eyes, loss of appetite and thirst. These symptoms appear nearly simultaneously; but those of fever are the ones which define most precisely the beginning of the disease, and influence most strongly the character and intensity of the prodromal symptoms. The temperature increases rapidly, and generally continuously to a considerable, or at least an appreciable degree (102°-104° Fahr.), rarely remaining under 102°; its course therefore is very different from that of the latent period, if perchance an increase of temperature should occur in the latter. The height of the maximal temperature, attained during the evening hours of the first day of the disease, enables us to determine in some measure, in primary measles, the intensity also of the later symptoms of the disease. As a rule, on the following morning, that is, on the second day of the prodromal stage, a considerable remission of the fever takes place, and the temperature frequently becomes even normal; or, exceptionally, it remains elevated for twelve or even twenty-four hours longer, and is then followed by the remission for the first time. To this febrile initial stage, which lasts on an average one day, succeed two days of usually very slight fever. On the first of these—the second day of the disease—the elevation of temperature is especially moderate, and in mild cases can even be entirely absent; this absence of fever, however, will not last beyond the morning hours of the following, the third day (when even a lower temperature than twenty-four hours previously may be observed), while at evening a more or less marked increase is rarely wanting. During this interval of more or less complete absence of fever, the other general symptoms usually subside, the natural vivacity returns, and even the appetite may be restored, though both may have remained normal from the beginning, owing to the mildness of the fever on the first day. The whole prodromal stage may thus escape the observation of the parents, who then assert emphatically that the disease first began with the outbreak of the exanthem, a supposition based upon error, as can be shown by accurate observation in each individual case. The catarrhal symptoms, however, generally increase during these days; the conjunctiva is reddened, the lids are swollen, the eyes shun the light and smart, and there is an increased secretion of tears. The nose seems stopped up, and begins by degrees to secrete mucus; sneezing takes place, and epistaxis may occur, rarely to such an extent, however, as to call for active interference. If the catarrh is severe, the face becomes œdematous; itching of the skin appears, and even an urticaria. The cough, at first dry, manifests itself usually on the first day, and becomes subsequently worse, assuming frequently a peculiar, harsh sound, though rarely so convulsive as in croup or whooping-cough (Weil). According to the severity of the catarrh the voice becomes thick and rough, or even quite hoarse. At times even sore throat may occur, the usual slight redness of these parts increasing to actual inflammation, the tonsils becoming swollen, and angina faucium with its sequelæ taking place. Weil observed parotitis and pleuritis during the prodromal stage. Gastric and intestinal symptoms, on the other hand, are either absent or moderate in the prodromal stage, with the exception of some loss of appetite; frequent vomiting is especially rare. Slight diarrhœa sometimes occurs. Frontal headache is frequent on the first day, if the fever is high, otherwise it is rare, and under the same circumstances a tendency to sleep often at first exists, but it disappears with the remission of the fever on the second day. Other slight nervous symptoms may be present, but the severe initial symptoms of febrile diseases in children, such as convulsions, for instance, are almost always absent. Edwards describes as peculiarities of the convulsions in the prodromal stage of measles: an extraordinary severity of the attack, with a duration, however, so short that the physician who is called rarely has an opportunity to observe it; the immediate return to consciousness after the attack without tendency to coma or sleep; the appearance of the attack but once, while other forms of eclampsia, especially the bad forms of the same in the eruptive stage, repeat themselves; the dilated pupils, which yet respond to the stimulus of light. Other authors do not allude to these peculiarities in the convulsions of the early stage, but report them as of the usual character. Such convulsions do not render the prognosis less favorable. Tüngel saw an epileptic, aged sixteen years, attacked by headache, clonic cramps in the arms and legs, and sopor; the twitchings returned on the following day to a less degree; three days later the eruption appeared, and thenceforth everything took its normal course. Wisshaupt noticed in a child of three years a similar influence of the eruption, upon the appearance of which the prodromal convulsions ceased.

The *eruptive stage* begins in normal measles on the fourth, or at the latest on the fifth day of the febrile period of the disorder. When this appears the remittent and intermittent character of the prodromal fever ceases, and instead we have the more marked and continuous elevation of temperature characteristic of the acme of the measles fever.

During the stage of eruption, and usually about thirty-six hours after its commencement, the maximal temperature is reached; this in normal cases corresponds with the maximum of the exanthem, or at least with the first stage of it. It is exceptional when the maximal temperature occurs near the beginning of the eruptive stage. The normal duration of the acme is from one and a half to two and a half days, corresponding conversely with the duration of the prodromal stage, so that the maximal temperature occurs with considerable regularity at the end of the fifth or sixth day of the disease. The period of the maximum of the exanthem, and with it of the maximal temperature, is more constant than the duration of either the prodromal or the eruptive stage, each of which compensates for the other. The time which elapses from the moment of infection to the maximum of the eruption (about fifteen days) appears to be still more uniform, and Panum has therefore employed this interval, or rather the shorter one, only to the beginning of the eruption, for the reckoning of the duration of the incubation.

The development of the spots, in other words *the eruption*, shows itself in milder cases at the beginning, in more severe ones only in the second half of the fourth day of the fever. Accordingly, the morning temperature of this day is usually higher in a mild case than in a more severe one, for it is the eruption which in this stage especially influences the course of the temperature. When the first considerable increase of temperature (as high, for instance, as 102.2° Fahr. and above) occurs in the morning, there is generally a further rise towards evening, followed by a moderate remission or none at all the next morning, and on the evening of this day (the fifth of the disease) the maximal temperature. When the beginning of the increase of the eruption occurs on the evening of the fourth day, there is usually the next morning little or no remission, and the subsequent course is the same as in the first case, except that the maximal elevation of temperature will take place on the evening of the sixth day. Often, however, regular remissions with subsequent exacerbations appear on two successive days, and the maximal temperature of the case is only reached in the second exacerbation. At another time the evening maximum may be absent and appear first on the following morning,—that is, where the eruption has begun early, on the morning of the sixth day. This is the state of things when the maxima of eruption and temperature coincide in cases of measles with a perfectly normal course. In a decided minority of cases, which are normal as regards the exanthem and the defervescence, the maximal temperature can appear somewhat earlier, at nearly the same time as the eruption, if not at its commencement, and the maximum of the exanthem may therefore be developed only after some decrease in the temperature, a state of things which often marks the occurrence of some complication. But the proposition cannot be advanced that the outbreak of the exanthem, rather than its maximal development, is the cause of the highest stage of the fever of measles. If this were the case, the temperature at the commencement of the eruption would not so often be only moderately elevated or nearly normal, whereas, when the eruption is nearly at its height, the temperature is always elevated. The maximal increase of the temperature during the acme is as a rule the most considerable elevation observed throughout the attack; only once in a while is it exceeded by the initial elevation on the first day of the disease. It occurs, as stated, usually toward evening. The highest point reached is on an average 104° (Fahr.), but often it may be as high as 105.8°, or even more, without the intervention of any cause outside of the exanthem.

The eruption makes its appearance at the time of the marked elevation of temperature on the fourth or the fifth day of the disease, and extends, during the continuance of the fever, over all parts of the body. At the maximal temperature, or near it, the spots are largest, reddest, and thickest, even confluent in places; they sometimes cause considerable itching. The face at this time, often independently of the eruption, is reddened, even somewhat sodden, usually only in consequence of the fever. If no abnormal complications occur, the affection of the mucous membrane of the mouth and throat remains at the beginning of the eruption in nearly the same condition as just previously, while at the time of the maximum of the exanthem it is often already somewhat faded. The inflammatory symptoms of the mucous membranes remain at the beginning of the eruption in about the same intensity as in the prodromal stage, but they soon decrease with the elimination of a more profuse secretion. Gradually the photophobia and the profuse lachrymation disappear, the cough becomes moister and less frequent, and is accompanied by a muco-purulent expectoration; the irritation of the nose, and, for the most part, the epistaxis, also cease. Other symptoms, however, sometimes become more severe at this period, as a consequence of the supervention of slight inflammations; thus, for example, the voice becomes rough or hoarse; there is difficulty in swallowing; dry or moist râles may occur throughout the chest; the gums may swell and become partly excoriated. Abdominal symptoms often appear at this time as a sign of a perhaps specific congestion of the intestinal mucous membrane, and *diarrhœa*, if not already present, is apt to occur, especially in little children and during summer epidemics, and in such cases the character of the stools and the intensity of the attack often remind one of cholera. Vomiting and severe colics more rarely ensue. The nervous symptoms, brought over perhaps from the prodromal period, increase with the intensity of the fever during the eruption, and afterwards definitely disappear when the remission takes place.

In the literature scarcely any mention will be found of the nature of the *urine* in measles, yet it is evident from even these scanty references that essential anomalies have been but rarely observed. Brown thinks, in opposition to Becquerel,—who has never met with albuminuria, even in cases in which the kidneys were congested,—that the urine may become albuminous when the eruption develops rapidly, and that this albuminuria generally occurs on the third day. It may be only a question here of a slight temporary amount of albumen occurring at the time of the highest fever, in consequence of the excessive increase of temperature, more rarely of a slight desquamative nephritis, in favor of which is the fact that now and then a profuse exfoliation of the epithelium of the urinary apparatus is observed. Thus Abeille twice found albuminous urine in measles, lasting for seven and eighteen days respectively. An essential participation of the kidneys, as in scarlet fever, does not occur in measles. In a patient affected a few years before with dropsy from scarlet fever, but decidedly free from albuminuria before the measles, I found a very transient though tolerably severe attack of the latter, during the eruption of measles, accompanied by a profuse separation of hyaline casts.

After the exanthem has reached its most intense development, the temperature, which at this time is at its maximum, begins also to sink, or—if it has abnormally reached its maximum somewhat earlier—to fall more speedily than before, with the fading away of the exanthem. This fall in temperature is more or less rapid, so that generally the normal condition is attained from one to one and a half days at most after the maximum has been passed; this frequently occurs on the morning after the evening of the maximum, more rarely first after from two to two and a half days. The most rapid fall takes place from evening to morning, exceptionally at some other time. When the course of the defervescence is rapid, the temperature sinks without any material interruption; when it extends over a day and a half or more, its course is remittent. From the evening of the maximal eruption to the next morning the temperature generally sinks considerably, frequently until there is hardly any fever, to perhaps only one or two degrees above what is normal. During the next period of exacerbation there is a new and temporary increase which never attains in normal cases the height of temperature of the previous evening; during this the rapidly blanching redness of the exanthem may increase again on all, or only the lower, parts of the body to a varying extent. Soon afterwards, in the beginning of the night, the further fall of the temperature commences, and by the following morning at the latest this is normal and continues so subsequently, provided no intercurrent disturbance occurs. Subnormal temperatures even may be present in the first days of convalescence, at other times the same days show trifling elevations, but only of tenths of a degree, or vacillations may occur between these two limits. These slight anomalies, occurring as a rule only when the local symptoms have been well marked, are of no importance, and give place in a few days to a perfectly normal temperature. The usual slight temporary desquamation of the skin begins at this time, if at all, and a similar, less noticeable process takes place upon the mucous membranes. The other symptoms of the disease abate in normal cases after the disappearance of the fever, and a slight susceptibility to external influences remains for at most but a short time. The mucous secretion from the nose and air-passages, the affection of the eyes and intestines, all cease by degrees, the appetite returns, sleep becomes less and less disturbed by catarrhal symptoms, the strength increases, the children wish to leave their beds, and may—at the end of three weeks (in simple measles), rarely after a shorter period—be considered, except where the disease has been very severe, as recovered. Yet they need for several weeks subsequently unusual care and watching.

Anomalies of the Course.

The normal course which has been described is that of the majority of the cases of mild epidemics. But even in these there is no lack of cases with an anomalous course, and these may in more severe epidemics constitute a much higher proportionate per cent. or even the majority.

The anomalies may pertain in part to the course of the measles in general or to that of single stages, in part to the development of the individual localizations of the process of measles. The last are much influenced by complications which usually appear also on other parts than those normally affected by the measles, and which are therefore capable of entirely changing the general appearance of this disease.

The abnormal forms of the course as a whole are of two kinds: the essentially *mild* and the essentially *severe.*

The two most important mild forms are *measles without catarrh* and *measles without exanthem.* Cases of the first sort occur now and then in every epidemic of measles, and attack more often the younger children, who have less individual susceptibility, than the more advanced or adults. They are generally also accompanied by less fever. Such cases destroy the susceptibility to measles no less than an attack running a normal course, for these are genuine cases of measles, though of the lightest form and imperfectly developed, but otherwise normal. They must not be confounded with rubeola, which has a peculiar course, and which certainly may occur sporadically, as also may mild measles without catarrh; generally, however, it appears, like the measles, in epidemics. The reports of an epidemic occurrence of light cases resembling measles in young, or more particularly in older children, must be received with suspicion, it being highly probable that the disease was rubeola. Rubeola affords no protection against measles, and furnishes an explanation of many cases of ostensibly secondary occurrence of measles in the same person. Moreover, it is only the mildness of the general course which gives to such cases of measles a certain resemblance to rubeola, and not the absence of mucous membrane symptoms, which, as a rule, are also present in rubeola to a moderate degree.

Measles without exanthem (morbilli sine morbillis) can have a varied course. This form of the disease may be diagnosticated in persons previously unattacked, if, in a single case, during an epidemic of measles, the characteristic mucous-membrane symptoms together with fever appear and become exactly as much developed as in measles with an exanthem, so that one has ground for assuming that this symptom alone is lacking from a normal course. The number of the well-established cases which manifest the tolerably characteristic deportment of the mucous membranes above described, is probably small; it may be assumed that the diagnosis of measles without exanthem is more often made than justified. Cases at times occur, likewise in persons previously unaffected, where the disease runs a usual course up to the time of the eruption of the exanthem, but then ceases without manifesting the exanthem and the fever of the eruptive stage. Such cases are therefore distinguished from other cases with irregular fever by the presence of the first half of the regular fever of measles. Whether morbilli sine morbillis was rightly diagnosticated in those cases, in which there occurred subsequently an unmistakable attack of measles, and in which, therefore, it must be assumed that the susceptibility to the disease had not been destroyed by the first attack, is an open question. It also appears to me rather doubtful whether, in ostensible cases of measles without eruption, where subsequently well-marked desquamation is said to have been present, the exanthem really was absent. I have never seen a convincing case of this sort, and such an occurrence would also conflict with the circumstance that the intensity of the desquamation is in general in proportion to that of the exanthem. And yet the occurrence of desquamation without an exanthem in the closely allied scarlet fever has been so often asserted as to hardly admit of any doubt, and may therefore be also assumed in measles, especially since observers like Seitz support it.

Salzmann had described as a striking anomaly of the exanthem the occurrence, during the epidemic of 1861 at Essling, of bluish, smooth, round, elevated spots, from the size of a nickel cent to that of a half-dollar, which took the place of the eruption, though accompanied by all the other appearances of measles, and at one time changed to the normal exanthem subsequently, at another remained unchanged until the end of the disease. These spots were due in Salzmann's opinion to an exudation in the subcutaneous cellular tissue; I should prefer to consider them as forms of urticaria.

An *anomalous* or a *rapid* and *virulent course* of measles appears usually with, rarely without, the appearance of a hemorrhagic diathesis.

True hemorrhagic measles are much rarer than the same form in variola. They occur even in little children, but are most common in the sick and debilitated, and are therefore generally secondary or only apparently primary. They must be especially distinguished from the previously described mild, hemorrhagic form, which differs only in the character of its spots from the perfectly normal form, and is very common in perfectly healthy and robust children. The oldest accounts of the malignant form are of little value, though by far the most numerous, for the "black measles" played formerly a great rôle. Doubtless its ostensible frequence is somewhat explained by the preposterous treatment of old times, but more particularly by the fact that measles and scarlet fever were included in one category, and malignant forms of scarlet fever were described as measles. Before or after the development of the eruption of measles, which, if present, usually fades quickly, hemorrhages ensue from vessels in all possible regions: in the skin, where at one time they give a violet coloration to many roseolæ, while at another they appear, independently of the exanthem, in the form of petechiæ and more extended ecchymoses; in the mucous membranes, where they manifest similar appearances or occur in the form of almost uncontrollable bleedings from the free surface (especially from the nose and kidneys, at times from the bronchi, and in women from the intestines and uterus); finally, in the parenchyma of organs, in cavities, and in the cellular tissue. In addition, we find as a rule important parenchymatous changes of organs. The cause of this disturbance of the normal course of the process must be sought in an alteration of the blood occasioned by the contagion, in consequence of which the blood-cells are largely destroyed and the normal composition of the blood becomes changed. Blood so changed is no longer capable of supplying sufficient nourishment to the elementary constituents of the organs; delicate changes in the vascular walls ensue, which may be the cause of extravasations from the intra-vascular pressure. Death generally occurs, before any considerable anatomical changes of the organs have taken place, a few days after the appearance of symptoms of the hemorrhagic diathesis; more rarely hemorrhagic inflammations and infarctions of single organs are present.

Where severe acute or chronic disease is present, the infection alone, without the production of hemorrhagic diathesis, may so shatter an already debilitated constitution as to cause death in a few days, and that, too, without violent fever and without any considerable local affection.

The prognosis is somewhat more favorable if the illness is prolonged. The anomaly can commonly be recognized at the very first from the great intensity of the scarcely remitting, and therefore untypical fever, which during the outbreak of an imperfectly developed, discolored, livid exanthem, goes on even increasing. Great weakness soon results; the pulse becomes small, and, for measles, uncommonly frequent, the tongue dry, the gums and lips covered as with soot, the abdomen distended, diarrhœa sets in, the catarrhal affection attacks even the finest bronchi, and *broncho-pneumonia* appears. The debility increases, frequent collapses occur, and death takes place in the second week, unless within a very few days the fever moderates;

recovery is only to be expected when, in a very moderately developed pneumonia, the remissions increase, the exacerbations by degrees diminish, the intensity of the fever lessens, and the nutrition and strength improve. The appearance of patients during the fever resembles in some measure that of typhoid patients, especially when the respiratory symptoms are not prominent, and this modification of the course of measles is therefore also called typhoid measles.

While the different stages, especially in malignant forms of measles, can be essentially altered or entirely obliterated, in other cases they may retain their general character and change only their form. The variations concern particularly the course of the fever and the deportment of the eruption.

Whether in anomalous cases the duration of the stage of incubation can vary, as has at times been asserted, is very doubtful. Most observations of this sort are ambiguous, and afford no opportunity for any positive decision. It may, however, in exceptional cases, be accompanied a part of the time by fever. An ephemeral fever during the stage of incubation has no significance, if it lasts but a short time, appears early, and towards the end either disappears altogether or moderates essentially. Should it, however, make its first appearance at the close of the stage of incubation, it obliterates, if intense, the commencement of the prodromal stage, and permits us to prognosticate subsequent anomalies in this stage also. Henoch relates a case of measles in which fever was present ten days before the normal eruption. It is possible, however, that a febrile incubative stage may not result from the infection of measles, but from some casual affection.

Essentially abnormal variations in the prodromal stage are often the results of the development of unusually intense local disturbances. Especially ominous is every marked access of fever at this period, with the exception of that on the first day, which is to be considered normal unless the increase is very considerable. Such unusual accessions of fever can immediately succeed the initial stage, with no interval, but can also, after a brief remission, characterize the end of the second day and the days following. The eruption, under these circumstances, is often accompanied by even more marked and sometimes extraordinary fever, but sometimes also by an unusual and anomalous diminution of it; both give rise to complications. Thus Politzer observed a prodromal stage lasting eight days, with typhoid symptoms and great depression, without high fever; the symptoms of prostration disappeared with the outbreak of a profuse exanthem. In another case the fever lasted four days with great prostration, but without any local manifestation; on the fifth day symptoms like cholera appeared, with great collapse; on the sixth, remission of these and the eruption, and with this the end of the previously anomalous course. Most complications originate, at least in previously healthy children, in an eruptive stage with fever, which succeeds a prodromal stage either normal, or from its fever and long duration abnormal. Again, the eruptive stage may be normal, and the period of defervescence be the first to manifest the existence of any disturbance, in that it is either unusually protracted by repeated exacerbations, or is immediately interrupted by a new and continued elevation of temperature. Such disturbances in temperature occur less frequently during the desquamative stage or convalescence, after the normal expiration of the fever of measles. The great value of measuring the temperature in the study of measles consists in this, that by it better than by any other means, and especially much better than by merely regarding the exanthem, normal cases can be distinguished from abnormal ones, the occurrence of anomalies and complications can be defined, and their significance determined. The prognosis is favorable when the course is but moderately abnormal, and especially if the first appearance of a slight disturbance of the characteristic course of the fever does not occur till late.

The already mentioned anomalies in the character of the exanthem are of less importance, for even in very anomalous cases the eruption can be of such a character as to justify us in considering it normal. The exanthem is anomalous in that it may be absent altogether, or may appear in varying manner upon different parts of the body; the spots may appear at first on the abdomen or the extremities and spread from here to the other parts, or they may develop only on individual parts of the body (Niemeyer), *e.g.*, only on the face and trunk, etc.; again, they may be absent on certain parts which usually are severely attacked, as for instance the arms or the neck (Neisser); or they may only appear upon one side (for example, after hemidrosis, only upon the perspiring side); finally, they may not appear on paralyzed parts (as, according to Hebra, in paralysis of the lower limbs from spondylitis), or they may appear here to either a very slight or a very marked degree. The same variations may also be observed in regard to the size of the single spots, which may be very small or so large as to be universally confluent; or in regard to their color, which may be unusually dark, hemorrhagic, or pale, and very different on different parts of the body; or finally in regard to their duration, which is exceptionally unusually long, or may be quite short. The short duration of the eruption was formerly thought to exercise a great influence upon the character and course of the disease, especially when complications were developed, since it was held that the retrocession of the former must be regarded as the cause of the latter; careful investigations have now shown that the complication is usually present before the exanthem disappears, and therefore cannot be occasioned by the disappearance of the latter.

The eruption in secondary attacks of the disease is usually anomalous.

Mention must also be made of the rare cases of a relapse of the eruption, which occurs after the commencement of retrocession, and is associated with a return of the fever; in these cases the spots appear on parts of the skin, which up to that time had preserved their normal appearance. As these relapses are usually of short duration, we are justified in including them under the anomalies.

Lewin, while describing a similar occurrence in scarlet fever, mentions incidentally a singular kind of anomalous course in measles, in two boys sick with the disease. The eruption appears to have suddenly ceased without causing any perceptible detriment, and after two weeks to have returned; furthermore, while in one case the disease ran a normal course, in the other this only occurred after a second equally harmless interruption of several days. Such a course is perhaps a parallel one to many of those relapses appearing after the first eruption, also to Trojanowsky's recurrent form of measles.

Complications.

Measles may be said to be complicated when the usual concomitant manifestations or other morbid processes are so intensely developed that they constitute the most prominent feature of the disease. The more important complications always essentially retard the recovery and thus acquire, in proportion as the characteristic symptoms of measles recede, more and more the significance of independent lesions. The frequency of their occurrence varies in different epidemics, and the form which they assume is due partly to the character of the epidemic, partly to individual peculiarities,—whether these consist of individal morbid predispositions, or of unfavorable external circumstances.

The skin becomes at times not only the seat of exceptionally numerous miliary vesicles, developing upon the customary papules of measles, but may even show a well-marked and profuse formation of pustules. Coincident chronic cutaneous eruptions are said to cause frequently an unusually intense desquamation. Single bullæ and eruptions of urticaria, particularly in intense exanthems, are no rarities. Erythema occurs at one time at the beginning of the disease with the initial fever, at another at the height of the eruption, especially if the exanthem is marked or the patient kept too warm. Meyer-Hoffmeister observed a scarlatinous erythema even during convalescence; Hauner frequently saw one resembling an acute lichen. It may be more or less intense and diffused over the whole body, or limited to a single region. Thus, for example, Gerhardt mentions having seen upon the femoral triangle a prodromal exanthem which preceded by a day the general eruption. In the eruptive stage herpes facialis is not rare. I have observed at this period zoster femoralis; Krieg and Löschner pemphigus bullæ; Thore, pustules of ecthyma; Salzmann, impetigo and urticaria (?); others later, furunculosis. Lafaye describes two cases of sphacelus which ended in recovery after the fall of the superficial slough, and were perhaps caused by the irritating and profuse conjunctival and nasal secretion; in one of these cases small, dark gray sloughs with red borders appeared on the sixth day after the eruption of measles, on the right ala of the nose and on the upper lip, and soon afterwards also upon the œdematous cheeks and eyelids. In connection with severe affections of the skin, abscesses and phlegmonous suppuration in the subcutaneous cellular tissue have been described, and Heslop has even seen emphysema of the skin after gangrene of the mouth.

More important are the complications involving the mucous membranes, the conjunctiva, the nasal mucous membrane, that of the throat and of the upper air-passages. Special mention must be made of the intense phlyctenular inflammations of the conjunctiva, with their consequences: profuse blennorrhœa, abscesses, keratitis, and iritis, as they occur in scrofulous children, especially those whose eyes have been previously diseased; also rapid keratomalacia. Furthermore, under similar conditions, occur severe inflammations of the nasal membranes and excessively profuse epistaxis, the latter, more especially in the prodromal stage and during the commencement of the eruption, in which case extensive bleeding produces fading of the exanthem, while moderate bleeding acts rather favorably. Abnormally severe affections of the mucous membrane of the throat also occur. Thus Blanckaert found a grayish-black coloration of the mucous membrane of the throat and upper part of the larynx, without softening, in the case of a child which was attacked by measles during a whooping-cough, and exhaled a foul breath for some days before it finally died from pneumonia. The larynx is often especially severely affected. The tone of the cough in measles is usually, at least at the beginning of the disease, rough and barking, and somewhat spasmodic; the attacks are frequent, with only short intervals, often interfering seriously with sleep; later on, the cough becomes less frequent, looser, and more moist; the voice, however, usually remains clear, and the respiration undisturbed. The case is different in more severe affections of the larynx, such as have been found more particularly in cases that have proved fatal from any cause, but also in those terminating in recovery. Barthez and Rilliet found, in nearly half of their autopsies of patients who had had measles, ulcerations and erosions of the mucous membrane of the larynx, especially upon the vocal cords; Hauner also found the same thing. These ulcers, according to Gerhardt, who observed them during life, occur preferably upon the posterior wall of the larynx, and are due partly to the follicular swelling characteristic of the catarrh of measles, partly to the mechanical irritation of the loosened mucous membrane produced by the frequent motion from coughing. They appear at times even in the prodromal stage, more frequently however, during the eruption, and occasion quite severe laryngeal symptoms, which are often designated as pseudo-croup. The cough is here dry and rough, exceedingly frequent and spasmodic, almost incessant; the voice is often very hoarse; the larynx is painful in coughing, speaking, or swallowing, and is the seat of a burning sensation; a rough, whistling, respiratory murmur is audible on auscultation, and often even at some distance; at times, especially with violent, incessant, irritating coughing or when drinking, there occur suffocative spasms and a painful sense of oppression (Mertens). Such attacks are specially induced by the presence of inspissated and very irritating deposits of mucus upon the laryngeal mucous membrane. The more violent symptoms usually moderate when the eruption reaches its full development, though the hoarseness and the barking spasmodic cough may remain for some time before the latter gradually becomes looser. These consecutive manifestations of intense laryngitis are distinguished from true croup by the absence of the pseudomembranes and the lack of signs indicating stenosis of the larynx. The catarrhal affection of the larynx spreads usually also to the trachea, and even the larger bronchi may be moderately affected.

Capillary bronchitis, especially when affecting a large portion of the lung, is far more dangerous than the simple, though perhaps severe forms of laryngitis and tracheo-bronchitis; for not only does the swelling of the mucous membrane of the smallest bronchi in connection with the profuse secretion, produce severe disturbance of respiration, but these processes lead also very frequently to further affections of the parenchyma of the lungs. Marked bronchitis is not so apt to appear in the prodromal stage as affections of the larynx and trachea; it is more likely to occur during the eruption, or even not before the fading of the exanthem, and then regularly delays the defervescence, or may even postpone it until after the varying duration of a more or less severe feverish condition; its course is then usually very irregular. Like pneumonia, it attacks by preference the very young, poorly nourished, atrophic, and scrofulous children, and like it also proves to be in such cases an extremely dangerous complication. The sputa accompanying it are more or less thick, muco-purulent, and may contain streaks of blood of varying aspects (Karg).

Among all the dangerous complications of measles, pneumonia is the most frequent, although in different epidemics great differences exist. It appears, like capillary bronchitis, especially in and just after the eruptive stage, and increases the fever of this, where it was relatively moderate, in proportion to the extent of lung involved. With the exacerbation of the fever the intensity of the eruption can likewise at first increase, but usually it disappears sooner than its wont, and leaves no trace; this formerly was the main ground for the opinion that the cause of the complicating pneumonia was the "striking in" of the exanthem. For the most part the pneumonia of measles is catarrhal, more rarely croupous in its nature. Croupous pneumonia is marked by rather high fever, with few remissions and interrupted at times by pseudo-crises, further by its complete or essential limitation to one, usually a lower lobe of the lung, and by the absence or slight development of the signs of bronchial catarrh. The frequency of the pulse and of the respiration is usually much greater than it had been before with the simple measles; pain in the chest is a common symptom; the cough, however, is often not increased, though usually it occurs in spasmodic attacks, and its violence corresponds to the stage of measles in which the pneumonia appeared. After the pneumonia has run on for a week or ten days, the fever is wont, as in genuine primary pneumonia, to decline speedily, convalescence taking place in the usual, undisturbed manner. Broncho-pneumonia acts otherwise. This never occurs without intense catarrh or other inflammation of the bronchi, and is therefore marked by numerous râles at the points affected. As in the usual catarrhal pneumonia, so here do we find at the autopsy an intense redness of the mucous membrane of the bronchioles, cylindrical dilatation and filling of the same by a tough, muco-purulent exudation, more or less extensive collapse of the lungs, especially at the lower border of the lower lobe and throughout the posterior portions of the lungs, where these lesions are sometimes found symmetrically situated on both sides, emphysema of the front upper parts, subpleural ecchymoses, and finally inflammatory thickenings, which exist either as solitary, smaller or larger nodules, or as uniformly diffused infiltrations. While the collapsed places are perfectly re-expanded by inflation, and then, in consequence of the resulting injection with blood, show a bright, almost cinnabar-red color, in the infiltrated parts this is only the case with single small lobules; these parts, if the lesion is recent, appear of a dark brownish-red color, void of air, as a rule irregularly shaped, and uniformly tough and resistant. Some weeks later these masses are often of a pale gray color, poorly supplied with blood, without air, rotten and crumbling, and give off when cut a large amount of thin fluid resembling pus. A characteristic of broncho-pneumonia is the unsymmetrical progress of the infiltration, the result of which is that, where the disease has existed for some time, a cut surface shows parts infiltrated with pus alternating in motley confusion with others infiltrated with blood and serum, or simply collapsed, or finally even containing air. Catarrhal pneumonia attacks, as a rule, both lower lobes at the same time, especially their dorsal aspects; while in the upper only a few smaller foci are usually present. The final extent of the trouble is such that, together with numerous more or less clear râles, an at least moderate dulness and slight bronchial respiration are rarely absent. The fever varies in intensity with the severity of the disease. In light cases the pneumonia may cause a moderate increase of temperature immediately succeeding the acme of the measles, or soon following it, and of short duration, its course strongly resembling the defervescence by lysis. In severe cases, however, the course, in regard to its intensity, often manifests a great resemblance to that of croupous pneumonia, and is distinguished from this only by its long duration and its slow, frequently interrupted defervescence, corresponding with the varying and irregular course of the local affection. The pulse is apt to be unusually frequent, but the other symptoms present no special contrast to those of a common broncho-pneumonia. Death occurs—especially with very young children, and then usually in the first week of the pneumonia—partly from the intensity of the fever, partly from the carbonic-acid poisoning, due to the rapid spread of the process and the absence of expectoration. The children become somnolent and pale, the extremities cool, the pulse constantly smaller and more rapid; the face and mucous membranes soon become markedly livid, the cough becomes less frequent, loud râles are heard in the trachea, and finally, after protracted agony, frequently interrupted by very distressing paroxysms, death takes place; or recovery may slowly ensue, and then often only several weeks after the beginning of the pneumonia.

Steiner and Neureutter observed gangrene of the lungs with measles in two instances. Both cases ended fatally.

Noteworthy disturbances of the auditory apparatus, often overlooked, owing to the fact that the symptoms may not be strongly marked, very frequently occur during the eruption, and immediately afterwards, as a result of the more intense affections of the naso-pharyngeal fossa. They are, according to Wendt (personally communicated), seldom purulent, usually simple catarrhal processes, limited to the Eustachian tube (swelling and obstruction of it by secretion), or also extending to the deeper portions (mucous secretion into the tympanic cavity, with or without swelling of its lining membrane). Hearing can thus be diminished by the mere accumulation of secretion, or it may be more markedly so from swelling of the mucous membrane in different ways, while in other cases, in which the affection of the tubes is more prominent, the hearing will vary at times. The exudation and swelling are sometimes preceded by pain; severe pain usually also precedes the mechanically produced perforation of the membrana tympani, this lesion being due to the pressure of the profuse secretion accumulated in the middle ear, and being followed by immediate abatement of the suffering. A more or less perfect recovery is tolerably frequent, and is due in favorable cases to the drainage of the secretion by means of ciliary motion through the gradually increasing calibre of the Eustachian tubes, to the retrogression of the swollen mucous membrane, and to the closure of any existing aperture in the membrana tympani.

The mucous membrane of the digestive canal can also become the seat of important complications of measles. Simple stomatitis (Weil, Dusével, Thore), also the aphthous (Hartmann) and ulcerative (Pank) forms, and glossitis are rare complications, though gingivitis is somewhat more frequent (Dusével). Mertens observed upon the lips, gums, and tongue a thrush-like pseudomembranous affection, which spread rapidly and occasioned in very young children such rigidity of the tongue that they could not nurse. Erichson saw in the prodromal stage, and also during the eruption, marked increase of the saliva; Weil, stomatitis; he and Heyfelder, also parotitis. Angina morbillosa is by no means rare, various authors to the contrary notwithstanding. Considerable parenchymatous tonsillitis, clearly recognizable by the difficulty in swallowing, may occur before the eruption, as an adjunct to the affection of the pharyngeal mucous membrane, already well developed in the prodromal stage or during the existence of the exanthem. Severe gastric affections are rare, but intestinal catarrhs of varying severity are quite frequent, and it is not possible to specify exactly where a light diarrhœa ceases to be merely that and becomes a complication; for it is probable that in measles the intestinal mucous membrane participates normally in the general congestion of the skin and superficial mucous membranes. Thus, the diarrhœa begins very frequently in the prodromal stage, or on the first day of the eruption, not so often during the same, and least frequently during the stage of retrogression, though these last cases are apt to be the most severe. The diarrhœa begins with or without previous indigestion, is painless or at times associated with colic, and passes rapidly and harmlessly away; or, especially after laxatives have been used for preceding constipation, it may last with great violence for even a week or more. In the case of little children, in whom, moreover, these intestinal complications are most frequent, death may ensue even in mild epidemics, and this occurs especially under the influence of the heat of summer, with gradual disappearance of bile from the stools and the appearance of even true choleraic symptoms. Or again, if the large intestine is more especially attacked, a dysenteric, bloody, mucous character manifests itself in the dejections, with tenesmus. Now and then, however, we hear reports also of malignant epidemics, in which even in winter (Kapff), and in adults, a fatal result was due chiefly to the affection of the intestines, or where the same caused at least great danger to the life of the patient. As a rule the affection of the intestines, since it rarely depends upon marked anatomical changes, heals soon after the fading of the exanthem, upon the duration and course of which, as upon the similar conditions of the fever, it exerts no influence except when it appears unusually early and produces by its great intensity a cholera-like collapse. The unfavorable influence exerted by the diarrhœa of measles appears to depend especially upon the fact that it diminishes the energy of the body, and renders it less able to resist the invasion of further complications. Besides the epistaxis, hemorrhages also occur in the prodromal stage and later from the anus and even from the kidneys and genitals, the last of which possess, however, of themselves no unfavorable prognostic significance; they appear during the menstrual period or at other times.

Measles differ from scarlet fever in this, that affections of other organs not essentially implicated in the process of measles, especially inflammations of important internal organs, so also of the serous membranes and of the joints, are tolerably rare. Yet now and then affections of various kinds occur, especially inflammations during the eruptive stage; they are mostly without influence upon the exanthem, but effect alterations in the course of the fever and prevent the rapid defervescence of the measles.

Conspicuous among the affections of the nervous system are: Meningitis (Spiess, Voit, Meyer-Hoffmeister, Kellner, Constant, Löschner, Thore, Bufalini, Krug); meningitis with tetanic and cataleptic rigidity of the limbs (Mettenheimer); spinal meningitis (Franque, Rilliet); hyperæmia of the brain (Geissler); meningo-encephalitis (Hannon); encephalitis (Rilliet); fatal "cerebro-spinal affections" (Barbieri, Mayo); apoplexy of the brain (Spiess); hemiplegia sinistra and paralysis of the left foot alone (Reichard); meningitis tuberculosa (Monti, Mayr); hydrocephalus acutus (Kronenberg, Hayden, Weil, Pfeilsticker, Heyfelder, Schallenmüller); hydrocephalus chronicus, augmented by the outbreak of measles, and consequently fatal (Heinecke); thrombosis of the cerebral sinuses (Faye, Routh); muscular paralysis resulting from an affection of the spinal marrow (Holmes Coote); universal paralysis (after gangrene: Bourdillat); paralyses and contractions (Hennig); mental disorder (immediately after the eruption: Mugnier); transitory mania and paralyses (Christian); delusions about persecution in the delirium of the collapse (Weber); severe cephalic symptoms of varying nature (Neureutter, Lippe); great sopor (Hauner); coma (cured: Schepers; with ischuria vesicalis: Mettenheimer, Brown); various cases of disturbances of sensation, paralyses, contractions of the muscles of neck, spasms of the glottis (Zavizianos); chorea (Sibergundi, Böning); tetanus (with tubercles of the brain as also with ischuria: Simpson); tonic spasms (especially in the flexors of the extremities, rolling spasms of the head, two days after the eruption, accompanied by its disappearance: Pinkham); convulsions and eclampsia, usually with a similar effect upon the exanthem, at different periods of the same (Bartels, Posner, Bierbaum, Adet de Roseville, Trousseau, Carroll, Edwards, Kaufmann, Liverani, Weil, Brown, Bartscher, Jütting, Brachet, Espinouse, Fichtbauer); neuralgias of the face (Imbert-Gourbeyre), and elsewhere (intercostal neuralgia, with convulsions: Rilliet; neuralgic arthralgia of a lower limb, with immobility of the knee, as an abnormal prodromal symptom: Köstlin); strabismus (Bierbaum) immediately after the period of eruption.

Among the affections of the thoracic viscera are to be mentioned: "Inflammations of the heart" (Hennig); endocarditis (Martineau, West, Köhler); pericarditis (Berndt, Majer, Espinouse, Braun, Siegel, Mettenheimer, Heyfelder; according to Autenrieth this is frequent); furthermore, gangrene of the lungs (Bartels, Mayr); tuberculosis (Ziemssen); pneumothorax from abscesses of the lungs (Barthez and Rilliet); pleuritis (Günzburg, Simpson, Salzmann, Monti, Voit, Rilliet, Berndt, Kellner, Spiess, Abelin, Trousseau); all these disturbances with or without the most usual complications: capillary bronchitis and broncho-pneumonia.

Nicola saw aphthæ spread from the buccal cavity over the whole œsophagus to the stomach. Further complicating affections of the abdominal viscera are, apart from those already mentioned, for the most part only catarrhal affections of the intestinal mucous membrane producing moderate diarrhœa: enteritis (Espinouse, Kellner, Luithlen, Lees, Rösch, Thore, Abelin); dysentery (Carroll, Kapff during a winter epidemic, with retrocession from the respiratory organs, Daniell, Rufz); intense neuralgic colic (Kapff, Meyer-Hoffmeister, Fricker); intestinal bleeding (Rilliet); ascites, without anasarca, with albuminuria (Zehnder); ascites (Pfaff); peritonitis (Simpson, Lees); tuberculous peritonitis (Spiess); "affections of dentition" (Bartscher). A moderate swelling of the spleen was proved by Clemens; a greater by Löschner, Lehmann, Huguenin; by the last, as also by E. Wagner, a swelling of the liver. Meyer-Hoffmeister several times observed icterus; Clemens the same on the cadaver, the liver being fatty and full of blood, and pneumonia also existing; Mettenheimer, icterus in the prodromal stage; Hennig records granular formation in the liver, and calls the bile diminished, yellowish-brown, viscid.

Of the affections of the genito-urinary apparatus and their consequences, are worthy of mention: Simple albuminuria, which, according to Mettenheimer, who saw it in a girl of seven months, is frequent in many epidemics (Vallon, Espinouse, Abeille, with anasarca; Kaurin); anasarca, without albuminuria, from obstruction in consequence of capillary bronchitis (Trousseau); anasarca without regard to albuminuria (Dubini, Hannon, Jütting, Seidl [especially with chronic diarrhœa], Sibergundi, Nicola, Billard, Rilliet, Liverani, Löschner, Hauner, Schott, Capuron, Becquerel, Barbillier); anasarca with intermittent albuminuria (Denizet); hydrops with and without albuminuria (Lombard, according to Rilliet, Kennedy, Flechner, Pfaff, Zehnder, Weil, Biett, Duchek); congestion of the kidneys (Becquerel); parenchymatous nephritis (Geissler, Röser, J. Frank, Rilliet, West, Kjellberg, Lehmann, Müller [fatal from uræmia], Bouchut, Malmsten [three to four days before the eruption], Spiess, Hauner, Thomas); morbus Brightii (Steiner and Neureutter, Zehnder); bleeding from the kidneys (on the second day of the eruption: Malmsten); anuria (Rilliet, Kolb); dysuria (Henoch, Rilliet); retention of urine (lasting forty-eight hours: Pfeilsticker, Köhler); profuse menstruation (Rilliet); abortion (Rösch); mild œdema of the genitals and extremities (Masarei).

Furthermore should be mentioned: Marked swellings of the lymphatic glands (Salzmann, Coley, Meyer-Hoffmeister, Mettenheimer); glandular inflammation and suppuration (Gregory, Rilliet); parotitis (Fichtbauer, Thore, Eisenmann, Bufalini, Battersey); abscesses in the subcutaneous cellular tissue (Barthez and Rilliet, Ottoni); sclerosis of the cellular tissue, fatal in a three-months' child (Salzmann); inflammation of the ankle (Meyer-Hoffmeister); synovitis of the knee (Köhler); coxitis (E. Wagner).

Finally, of constitutional diseases there immediately followed measles: intermittent fever (Ziemssen, Meyer-Hoffmeister); acute rheumatism (Salzmann); hæmorrhagic diathesis (Trousseau); morbus maculosus Werlhofii (Mettenheimer, Masarei); rachitis (Mettenheimer).

More important complications of measles are the following general diseases: Acute miliary tuberculosis; the tubercles appearing specially in the lungs and the membranes of the brain. The disease at times immediately follows the exanthem, and runs a fatal course in a few days or weeks, appearing under the form of an intense bronchitis and with cerebral symptoms like those of acute hydrocephalus, but with a high fever and frequent pulse; the very sensitive skin is in such cases frequently the seat of erythematous processes. Diphtheria is far more rarely associated with measles than with scarlet fever; it affects, by preference, the organs of the throat: at times the pharyngeal portions (Fichtbauer, Steinthal, Förster, Bartels, Hauner, Voit), including the back nasal passages (Steudel); at times the larynx and trachea (Abelin, Spiess), and its course is that of simple primary diphtheria. The disease can remain confined to the tonsils and other pharyngeal parts, or spread from here to the air-passages and even into the bronchi, or begin at the larynx and extend upwards and downwards, or affect this alone, and so on; it can, if of moderate extent, lead to slower or more speedy (Rothe) recovery, while it usually proves rapidly fatal if the air-passages are early implicated. Diphtheria generally appears at the acme of the eruption of measles, or soon after; is in the light forms, as, *e.g.*, croup of the tonsils, without influence upon the character of the exanthem, while in severe cases, when the air-passages are greatly affected and the appearance of the diphtheria is temporarily coincident with that of the exanthem, the latter becomes changed as with broncho-pneumonia, which then, moreover, generally sets in as a further complication. The normal type of the course of the fever is affected by every severe complication, and so, when diphtheria complicates the disease, the fever is usually heightened and protracted. The diagnostic symptoms of the diphtheria are furnished, as in the common form, by the inspection of the pharynx, by the cough and hoarseness, and by the consequences of the stenosis of the air-passages; and these pseudo-membranous affections of the organs of the throat must be clearly distinguished from the already-mentioned severe inflammations which are not pseudo-membranous, although about equally dangerous. Diphtheria of other organs than those of the throat is very rare in measles. Diphtheria is recorded of the eyelids (Mason), of the conjunctiva (Hauner), of the prepuce (Schreiber), and of the female genitals. Ravn and Aarestrup saw a fatal case of secondary diphtheria of the nose, œsophagus, and eye-lids, in consequence of which parenchymatous nephritis had also resulted. In debilitated, scrofulous, and rachitic children, rarely also in those previously healthy, measles can give rise to ulcerous and gangrenous affections. The ulcers manifest from the first a discolored base, whether they have proceeded from a catarrhal and apparently simple affection, or from an originally suspicious one (gangrenous bullæ, branny infiltration). They occur especially in the buccal cavity and on the genitals of boys and girls (noma). In the former case they take their origin from the gums, the lips, the mucous membrane of the cheeks, the cavity of a tooth, etc., and can go on uninterruptedly in their course while they destroy very considerable portions of the skin of the face and of the base of the tongue, even of the muscles of the tongue as far as to its extremity ; or they may cause at least a partial separation of the attachment of the tongue and consequent difficulty in swallowing (Bartels), or even partial exfoliation of the nasal bones (Huxham), and of the jaws (Huxham, Sadler, Bartels, Bresseler), or at least loss of the teeth (Bresseler). This noma arises for the most part only after the fading of the exanthem, and therefore does not influence its course; it, however, generally occasions more or less violent fever with its consequences, loss of appetite and diarrhœa, and respiratory symptoms. Death does not always occur; where the course is relatively mild, the patients can recover with disfiguring losses of tissue (Bentley described a case where such a stenosis of the mouth was formed that only one finger could be introduced, and then only with difficulty). At the frequent autopsies one finds the extremest anæmia, atony and emaciation, gangrene of the lungs, and broncho-pneumonia, while the vessels of the gangrenous mass remain unchanged. Apart from the above-named anomalies of constitution, and residence in badly ventilated, overcrowded rooms, the misuse of many drugs, as for instance calomel, is spoken of as a special cause. Noma (gangrenous ulcer) of the genitals is more common with girls than with boys, in whom it may begin at the prepuce and progress as far as the navel (Bartels). Gangrene of the vulva is developed in the same way as that of the buccal cavity, and can destroy the labia, the vaginal entrance, the soft parts at the mons veneris, even the perinæum as far as the anus. The secondary results are the same as those already mentioned.

Gangrene may also occur after measles upon other ulcerating or eczematous portions of the skin, especially on the nasal alæ and the external ear (Mayr, Triboulet, Causit). Mayr observed gangrene of the forearm in caries of the radius; Faye describes a case in which gangrene spread from the finger upon the forearm; Battersey a similar one, also a case of gangrene on the lower lip; Faye still another, where some pustules which had arisen during the eruption produced gangrene and extensive destruction of tissue upon the loins; Thomas (of Paris) mentions the case of a child of two years, on whose nates extensive gangrene was developed. Carroll selects from a severe epidemic of measles at Sydney several cases of special malignancy, where on the face and thorax only a few dark blotches appeared, while upon the extremities single vesicles were formed, which rapidly increased to a large size, burst, and became gangrenous; the affection extended with such rapidity that sometimes within twenty-four hours the whole epidermis would be lost. Masarei observed upon the soles of the feet and the palms of the hands, during the desquamation, large bullæ, which burst, leaving obstinate, painful ulcers. In rare cases gangrene of the lung occurs.

Finally, in measles, appearances of scurvy occur now and then, especially in the buccal cavity, without giving rise to gangrene, and either with or without further symptoms of the hemorrhagic diathesis already referred to.

Sequelæ.

The sequelæ of measles are those maladies which arise in consequence of it, and remain after its termination as independent diseases, or which appear for the first time after the measles without being immediately due to any complications of the same. As co-operating toward this end in these cases may be mentioned all unfavorable influences present in the constitution of the individual (scrofulosis, rachitis, chlorosis, tuberculosis, and impoverished nutrition in general), or which have acted upon the patient from without (bad nourishment, damp, badly ventilated dwellings, bad treatment and nursing). Especially important are the following: chronic diseases of the skin, pustular eruptions over the whole body (Braun), gangrene of the skin (on the neck: Gruel), furunculosis, abscesses; ozæna, sometimes with, sometimes without the elimination of a thin, offensive, more or less profuse secretion, in consequence of which eczema of the face usually ensues; chronic ophthalmia: sometimes blepharitis and the frequent formation of hordeoli, sometimes conjunctivitis, with especially annoying photophobia, keratitis, etc., in various forms, and at times with the worst results (*e.g.*, keratomalacia, according to Beger; paralysis of accommodation and consequent strabismus convergens, according to Coley; even capsular cataract); chronic affections of the auditory apparatus: according to

Wendt (personally communicated), formation of adhesions (immediate or from new-formed duplications of the mucosa) between the ossicles or membrana tympani and the walls of the tympanum, taking place while the parts are swollen and inflamed, and resulting in more or less diminution in the hearing; transition to the chronic form of catarrh of the middle ear, with tendency to hypertrophy and other alterations modifying unfavorably the physical properties of the mucous membrane; also suppuration with its consequences (otorrhœa). Gummers saw very frequently chronic enlargement of the tonsils after measles. Still further sequelæ are chronic catarrh of the mucous membrane of the respiratory organs, both of the larynx and trachea, and of the bronchi, of the most variable nature and intensity, sometimes with a character like whooping-cough (Bartels); asthmatic attacks (Eisenmann); chronic lung affections; pericarditis (Kellner); affections of the heart (Testa and others, according to Eisenmann); affections of the parotids (Eisenmann, Seidl, Schultze, Kellner); chronic inflammations of the periosteum and of the joints (Niemeyer); caries (Seidl). It is not uncommon for children, apparently recovered from measles, or convalescent, to be seized anew with difficult respiration, and after a longer or shorter duration of the new disturbance to even die, sometimes of cheesy pneumonia, with or without tubercles, sometimes from general miliary tuberculosis or tubercular meningitis, the causes of which, as it appears, must be especially sought in the cheesy degeneration of the swellings of the lymphatic glands occurring in the course of measles. The tuberculous bronchial glands in particular afford a frequent point of origin for tuberculosis of the lungs after measles; Pank observed tuberculosis of the mesenteric glands. The diphtheritic and gangrenous processes during and immediately following measles can induce a great variety of sequelæ: ichorous suppuration in the throat and subcutaneous cellular tissue, diphtheritic paralyses, affections of the lymphatic glands with their consequences, diseases of the kidneys, disfiguring cicatrices. Severe chronic intestinal diseases can result from intense affections of the small and large intestines in measles: chronic entero-colitis with wearisome diarrhœa, intestinal ulcers and stenoses, with ascites, icterus, and cardialgia

(Seitz), and their unfavorable influence upon the nutrition of the entire organism, whereby scrofulosis, rachitis, habitual anæmia, and other constitutional diseases can be induced. Stone in the bladder, according to Coulson, is also to be enumerated among the sequelæ of measles, in the case of children. Masarei describes a very acute febrile dropsy without albuminuria, which caused in eight cases, within a few days, the death of patients decidedly convalescent; in such cases he observed also "scurvy mostly in the form of purpura." Seidl reckons pleuritis and hydrops among the sequelæ of measles. Gley observed very intense "purpura hæmorrhagica" some days after the disappearance of the exanthem, together with scorbutic appearances in the mouth.

It is a very common experience that after epidemics of measles the children who have been affected are more prone to all sorts of attacks than at other times; they are anæmic, out of humor, dull, without appetite, etc. Among the severe acute diseases we should mention more especially the strikingly frequent appearance of croupous pneumonia for a period of several months after the conclusion of the epidemic, especially in winter and spring.

Secondary measles can exert various influences upon the primary disturbances. When the latter belong to the common complications of measles, they usually grow worse upon the appearance of secondary measles. Should these, for example, occur in the course of, or during convalescence from a pneumonia, this becomes worse or reappears, and in all cases recovery is delayed; so, too, a bronchitis is likely to become aggravated to a capillary bronchitis and broncho-pneumonia. Phthisical processes become speedily worse, and lead more quickly to death. Walz lost by suffocation a girl of five years, suffering from aneurism of the aorta. Children with chronic diarrhœa are usually made much worse by measles, and in general a previous gastro-intestinal affection predisposes remarkably to the diarrhœa of measles, *e.g.*, in teething children (Walz). The same explanation is undoubtedly to be given of the report of Polak, Mayr, and Weisse, viz., that children with measles are very often attacked by cholera, and usually in a very marked degree. The cause of diphtheritic and gangrenous affections, especially of the buccal cavity and of the genitals, must be especially sought in slight primary disturbances of these parts (hollow teeth, slight gingivitis, scrofulous leucorrhœa), and decidedly gangrenous affections of other parts have also a similar origin. Should measles, on the other hand, appear during a disease to which they do not usually give rise, they may favorably influence the course of the latter. Thus they have frequently exercised a curative effect, according to Rilliet, Taupin, Guersent, Rayer, upon chronic diseases of the skin; according to Behrend, not only in children, but also in the case of a woman of forty years, whose eczema of the scalp of three years' duration he saw disappear permanently after measles. Barthez and Rilliet saw chorea, epilepsy, and incontinence of urine of several months' duration healed by measles; furthermore, they observed that an anasarca after scarlet fever disappeared coincidently with the eruption of measles. Rilliet found that a chronic coxitis improved noticeably after measles. Weisse reports that measles, in the case of a girl suffering from convulsions, entirely removed this disease; Mettenheimer, that a boy suffering from nervous winking of both eyes lost this evil entirely during measles, though after several weeks it by degrees returned; measles, moreover, put a stop to a peculiar sort of nervous cough of three months' duration. Guersent noticed, with the beginning of the fever of measles, permanent relief from epileptiform attacks, which had appeared in consequence of a fit of anger, and of which the patient had had several daily for quite a length of time. Schmidt saw a girl of six years, who for a year had suffered from frequent daily convulsive attacks, which had so reduced her strength that death was expected, recover entirely owing to measles; he also treated a boy of five years, with a contraction of the lower extremities lasting for six months, in whom this disappeared as if magically with an attack of measles. Feith and Schröder van der Kolk report the case of a woman who for five years had been in an insane asylum with violent attacks of mania, which did not return after recovery from measles, and the woman was soon so well as to be discharged. Mombert and Michele mention the passing of lumbricoid worms, in consequence of measles, as a frequent occurrence. Hildenbrand saw an obstinate disease of the joints, which had been treated for three years in vain, heal after measles, of itself, in a short time; so also obstinate glandular tumors dispersed by it in the same way. Mettenheimer saw a caries of the tibia strikingly improve immediately after measles; Röser, a caries of the hand of a year's duration, in the case of a boy three years old, heal speedily after measles. According to Levy, an old gonorrhœa of the penis disappeared with the outbreak of an eruption of measles, which was immediately followed by varicella; after the expiration of this the gonorrhœa immediately returned; in another case after measles it did not return. Pank saw an obstinate ophthalmia disappear for a short time under the influence of measles.

Diagnosis.

The diagnosis of measles is based, first, upon the exanthem, secondly, upon the character of the mucous-membrane symptoms and of the fever, attention being also paid, in doubtful cases, to other attacks of this malady at the same place and time, especially such as may stand, as regards contagion, in direct relation to the case in question. The following should be remembered as diseases which, at definite periods of their development, frequently possess a striking resemblance to measles: rubeola, scarlet fever, variola, varicella, the different roseolæ, and typhus fever.

The eruption of rubeola can closely resemble that of measles; the points of difference are the indented character of the blotches of measles, the slight papular prominences upon them, the casual confluence upon the face, the more intense and protracted fever at the time of the eruption,—preceded, as stated, by a prodromal stage, while rubeola can run its course without fever—and the more violent symptoms upon the mucous membranes in measles. Many of the light epidemics of measles observed in former times may have been in reality epidemics of rubeola.

The eruption of scarlet fever occurs often in small spots, bearing some resemblance therefore to the similar form of measles, and eruptions of scarlet fever can even occur in the form of large spots, thus increasing the possibility of error. On the other hand, an almost universally confluent eruption of measles can exceptionally take on an appearance resembling scarlet fever. Points of distinction are afforded by the early eruption of the exanthem in scarlet fever, also by other concomitant appearances, especially the absence of coughing and sneezing in scarlet fever; the absence of early angina, of the scarlet-fever tongue, and of the marked swellings of the lymph glands in measles. The character of the fever aids our decision if it runs a normal course, since in measles it possesses a very characteristic curve. The so-called relapse of scarlet fever (pseudo-relapse) often possesses, according to my experience, great resemblance to measles. It is to be distinguished by the character of the fever, by the form and extent of the blotches, the difference from measles as to the character and site of confluence, the absence of the papules of measles upon the individual spots, as well as of the characteristic mucous-membrane symptoms, finally by the casual (coincident, but to be referred to the first outbreak of scarlet fever) affection of the kidneys, which is decidedly typical of scarlet fever. This resemblance of the relapse of scarlet fever to measles may have given rise to the common opinion that measles often immediately succeed scarlet fever.

Typhus fever often causes well-founded doubts in coincident epidemics of the two diseases. Its exanthem is by no means rarely papular and even hemorrhagic, like that of measles, and a catarrhal affection of the air-passages, especially of the trachea, is one of its usual concomitant symptoms (Pastau). In two of Rautenberg's children with typhus, the papular roseola was so thick that one could hardly help confounding it with measles; as a rule, apart from the fact that the fever and the course of the disease are different, the deciding symptoms against measles are the absence of or sparse eruption upon the face, the absence of catarrh of the nose and conjunctiva, and lastly, the marked swelling of the spleen. According to Naunyn, children with typhus may be attacked by nasal catarrh, conjunctivitis, and cough, and an exanthem perfectly resembling measles may appear after three days; while Kierski observed these mucous-membrane symptoms for the first time at the end of the first week of the fever. A correct decision may perhaps be reached by observing the condition of the palatal mucous membrane before the outbreak of the exanthem.

In variola the blotchy prodromal exanthem presents difficulties, though only for a short time, since by the next day the protruding papules remove all doubt. Moreover, there is the peculiar limitation of the spots in variola to certain parts of the body, the absence of the fine papules of measles, also of the mucous-membrane symptoms typical of measles.

The same is true of the spots at the commencement of varicella, at which time fever is also frequently lacking. Measles with large miliary vesicles may cause suspicion of varicella, and the latter, if the single vesicles are unusually small, and interspersed with profuse well-developed roseolæ, be mistaken for measles; the next day, however, will be sure to furnish typical proruptions.

The symptomatic roseola of other diseases, such as typhoid fever and cholera, is clearly defined by the rest of the course of the disease.

Measles and simple roseolar eruptions are distinguished by the partial confluences of the spots in certain regions of the body in the former disease; by the absence, in roseola, of injection of the palatal and pharyngeal mucous membranes, and especially by the absence of the concomitant symptoms of measles; finally, by the characteristic course of the fever in measles. It is very true that one is not likely to commit an error, if able to follow the entire course of the disease; but it is quite possible to err if, as is usual, one is called upon to make the diagnosis of a more or less rudimentarily developed exanthem, or of one which has only half run its course. In such cases measles are to be excluded, if the temperature is normal, if there is a vivid coloration of the eruption on the trunk, if the separate spots have a very smooth character, and if there are no symptoms on the part of the mucous membranes. If doubt still remains, it is often entirely removed by the absence of contagiousness. Epidemic, measles-like eruptions in southern lands (dengue, Malta fever, etc.) are sufficiently characterized by their peculiar symptoms. Roseolar exanthems produced by decaying straw are said to resemble measles very closely (Salisbury), but the statement that they afford protection against measles is not to be believed for a moment.

The darker complexion of the non-Caucasian races presents especial difficulty for the diagnosis of measles, as of other hyperæmiæ. We learn from the communications made by Pruner in regard to this point, that the exanthem in the brown-colored Abyssinians, Nubians and Fellahs appears in the form of irregularly indented, coppery blotches, and occasions a marbled appearance, while that of the negroes is distinguished by little vesicles, like lichen papules or miliary vesicles, or, according to Mauger and Rigler, by papules appreciable to the touch, which last fact, together with the symptoms from the mucous membranes and the furfuraceous white desquamation, was made use of by Roux in diagnosticating the disease in the case of Indians.

The diagnosis of measles without exanthem can be made with some degree of certainty only during an epidemic, and in cases where, the patients having been previously unaffected, the symptoms on the part of the mucous membranes are perfectly typical; one must regard the same factors in the diagnosis of measles in the prodromal stage, and must also especially notice in such a case whether some signs of the future exanthem are not present on the face and on the neck.

Prognosis.

The prognosis of primary and uncomplicated measles is thoroughly favorable, death from the severity of the infection alone being extremely rare. It is only owing to diseases during the course of which it is developed, or to the complications which are induced by it, that it becomes a disease the mortality of which is not insignificant, and in rare cases can even become excessive. With a typical course of the individual stages of the disease the result is always favorable; therefore all such deviations from the type as signify an aggravation of the trouble are unfavorable. In severe cases such deviations may begin even in the prodromal stage, though more frequently they first appear in the stage of eruption, and most commonly only when the exanthem has passed its acme. The anomalies most important to a prognosis are: unusually high fever and delay of its crisis; very profuse and vividly colored or anomalous exanthem; unusually intense affections of the mucous membranes; finally, complicating affections of internal organs, or some complicating general malady. Specially ominous are: high or increasing fever on the second and third days of the prodromal stage, also protraction of the same beyond the normal duration, therefore retardation of the eruption when the fever is high; intense exacerbations of temperature at the beginning of the eruption, with normal or anomalous prodromal stage,—generally forerunners of further anomalies; exceptional elevations of temperature (105° Fahr. and more in the axilla) at any period of the disease, even at the time of the maximum of the exanthem; finally, duration of the fever after the stage of eruption, in place of the crisis. This last behavior of the temperature indicates with great certainty in most cases the occurrence of complications of the most varying nature. Furthermore, the prognosis is modified when the eruption is anomalous: unusual sparseness and paleness of the same, with high fever; universal confluence or a hemorrhagic character of the spots; partial eruptions; beginning of the same elsewhere than upon the face, or anomalous diffusion over the body; premature and sudden fading or abnormally protracted duration of the eruption in the condition of most marked development. All severe complications can affect the favorable prognosis of measles, especially broncho-pneumonia, croup and diphtheria, intense diarrhœa, convulsions, particularly in the eruptive stage, also severe cerebral attacks in general, inflammations of internal organs, gangrenous affections, keratomalacia; many epidemics have become fatal from these disturbing influences. Profuse perspiration and severe hemorrhages are also unfavorable symptoms. The prognosis of secondary measles is unfavorable, especially if this, as is often the case, is at the same time complicated; but even without any complication the result of secondary measles is often fatal. Much naturally depends upon the nature of the original affection; patients are especially endangered who suffer from chronic thoracic affections; the same is true of the anæmic and poorly nourished, those who have intestinal catarrh or who are predisposed to disturbances of the brain (hydrocephalus and convulsions).

Healthy children between the ages of four and five have measles most lightly; younger ones, with the exception of the youngest sucklings, are often severely attacked. Teething children appear often to be attacked with special severity. I have several times seen them die with uncontrollable fever and severe nervous symptoms, and have discovered on the cadaver tooth-points just broken through. Measles is also a severe disease for older adults; they are rarely, however, attacked in these days; it is also severe for pregnant women, who readily abort, and for those recently delivered.

Death occurs rarely in the first, mostly in the second week of the disease; also later, according to the time of the accession of the fatal complication.

Those anomalies in the course of measles, which often interfere with a good prognosis, occur especially with patients in the first years of life.

According to Kellner there were, of 18 cases during the first year of life, 8, = 44 per cent., anomalous; of 61 during the second, 32, = 52 per cent.; of 84 during the third, 29, = 34 per cent.; of 168 during the fourth and fifth, 36, = 21 per cent.; of 204 during the sixth to tenth years, 43, = 21 per cent.; of 34 during the tenth to fifteenth years, 7, = 20 per cent.; of 11 during the fifteenth to twentieth years, 2, = 18 per cent.; of twenty cases above twenty years, 2, = 10 per cent. Spiess found the relative per cent. for the first year = 7 per cent.; for the second, = 42 per cent.; for the third, = 25 per cent.; for the fourth, = 16 per cent.; for the fifth, = 15 per cent.; for the fifth to tenth, and tenth to fifteenth, = 12 per cent.; for the fifteenth to twentieth, = 4 per cent.; anomalous cases of over twenty years gave 26 per cent.

These figures show plainly the diminishing tendency to complications with increasing age; it is only after youth is passed that an increase begins again. Anomalous cases are said to especially characterize the beginning of an epidemic (Kellner); there are proofs enough, however, of the contrary.

The age of the patients is, under all conditions, of the greatest influence upon the mortality of measles. Disregarding the fact that healthy and very young children (up to about the age of six months), probably from their feebler predisposition, are attacked very mildly, if at all, the rule may be laid down that measles are essentially dangerous only for young or very young children; that its danger decreases rapidly with accession of years, and in the late years of childhood is already at a minimum; in old people, who have, however, but little predisposition and are rarely attacked, the disease is again dangerous. Exceptions to this are not often reported.

Thus Schüz saw, in particular, children of from six to eight years die. In the Paris garrison, that is to say, among people between eighteen and thirty years of age, numerous fatal cases of measles took place in 1838, '39, '48, '49, '55, '60, due, according to Laveran, to the influence of vitiated hospital air. The following estimates can testify to the correctness of the rule given above: According to Schiefferdecker there died in London of measles from 1856–66: in the first year of life, 3,368; in the second, 7,606; in the third, 4,261; in the fourth, 2,247; in the fifth, 1,184; from 0 to five years, 18,666; from five to ten years, 1,076; from ten to fifteen years, 84; above fifteen years, 111; total, 19,937. So at Königsberg in six years: in the first year of life, 88; in the second and third, 157; from the fourth to the tenth, 115; from the tenth to the twentieth, 2; of older persons, none at all. According to Passow the absolute mortality from measles in Berlin, in 1863–67, increased up to the second year of life, at which point it reached its greatest height: 24 per cent. of all the deaths were in the first year, 31 per cent. in the second year. From the third year on it diminished, at first rapidly, then slowly, up to the thirtieth year, not constantly, however, since in the eighth and tenth years there was a slight increase, while from the twentieth to the twenty-fifth year no deaths took place. From the thirtieth to the thirty-fifth year the mortality again increased slightly; above thirty-five years there died only one person, aged sixty-two. According to Ranke there died in Munich (1859–68) 70 children under one year (out of 195), 119 at the age of from one to five years, 11 persons above fifteen years (out of 185 sick); the mortality of the first five years was therefore 94.5[1] per cent. In Würzburg it was, according to Voit (1842–71), for the same years of life about 93[1] per cent.; there died of 88 patients under one year, 21, = 23.8 per cent.; of 367 from one to five years, 15, = 4 per cent.; of 289 from five to fifteen years, 3, = 1 per cent. In the Vienna Children's Hospital there died, according to Monti, 1864–67, of 372 cases of measles the monstrous number of 98, of which 6 (out of 16 patients) were between six months and one year old; 70 from one to five years (out of 173 patients, namely, 35 patients with 21 deaths in the second year; 52 with 26 deaths in the third year; 47 with 13 deaths in the fourth year; 39 with 10 deaths in the fifth year); 22 from five to eleven years (of 183 patients there were 43 from five to six years, with 9 deaths; 38 between six and seven, with 6 deaths; 33 between seven and eight, with 4 deaths; 32

---

[1] These are probably typographical errors; 24.5 per cent. and 23 per cent. being intended.—TRANSLATOR'S NOTE.

between eight and nine, with 3 deaths); of persons above this age no one died. According to Geissler there died in 1861 at Meerane, out of 1,754 patients, 63; out of 18 under six months, no one; out of 99 from one-half to one year, 2; out of 221 from one to two years, 19; out of 264 from two to three years, 26; out of 226 between three and four years, 7; out of 204 from four to five years, 6; 1 each out of 187, 151, 144, of six, seven, and eight years respectively; of 227 older children (up to fourteen years) no one died. According to Spiess (Frankfort, 1860–61) the mortality equalled for the first year 8 out of 45 cases, = 18 per cent.; for the second, 15 out of 156 cases, = 10 per cent.; for the third, 9 out of 204 cases, = 4.4 per cent.; for the fourth, 3 out of 186, = 1.6 per cent.; so also for the fifth (4 out of 243); for the fifth to the tenth, = 0.7 per cent. (7 out of 954). According to Kellner there died at Frankfort, in 1858, 43 children of measles: 8 in the first, 18 in the second, 5 in the third, 4 each in the fourth and fifth, 1 in the sixth and seventh, 2 in the eighth year, above eight years no one. The influence of measles upon the mortality of different ages is very well shown in the epidemic of the Faröe Islands observed by Panum. During this, *i.e.*, in the first nine months of 1846, far more people died than ought to have done so in accordance with the average for the year, and of these, in the first year of life, nearly 3 times more, between one and twenty years the normal proportion, in the third decade 1.4 times more, in the fourth to the eighth decade 2.4 times, 2.6 times, 4.5 times, 3.9 times, 2 times more, between 80 and 100 years 1.5 times more. The chief portion of this excess of deaths was due to measles, which is therefore more dangerous the older the patients are; the decrease of the mortality in the oldest decades was due to the fact that only sixty-five years had elapsed since the last epidemic, and the oldest people were therefore for the most part no longer liable to attacks of measles, and therefore could of course not die from them.

Sex has no influence upon the mortality of measles: according to the reports, the number of deaths among boys at one time slightly exceeds that among girls, while at another it again falls below it.

The mortality of measles in general is as a rule slight, so that it may justly be reckoned among the least fatal of the infectious diseases.

Thus, according to Faber, there died in the epidemic of 1827–28, at Schorndorf only 1.8 per cent. of 2,100 cases; according to Geissler, from 1835 to 1869, at Meerane, only 2.1 per cent. of all the deaths among children were from measles, the severe epidemic of 1861 causing only a mortality of 3.5 per cent.; according to Ranke, in four epidemics at Munich, the mortality varied from 0.7 to 2.7 per cent.; in the Children's Clinic at Würzburg, there died, according to Voit, out of 851 cases of measles, from 1842–71, 39 = 4.5 per cent.; in Stuttgart, according to Köstlin (1852–65), 1.8 per cent.; the epidemic at Frankfort (1858), occasioned, according to Köstlin, a mortality of 2.4 per cent. Occasionally an epidemic is marked by special malignancy, owing to the occurrence of severe complications, which markedly increase the number of fatal cases. Thus, there died at the Children's Hospital in Würzburg, in the epidemic of 1863, 10.5 per cent.; in the Grand Duchy of Baden, according to Meier, from 1818–24, 5.4 per cent.; in an epidemic at Sydney, according to Carroll, 6 per cent.; at Leith, according to Brown, 9.7 per cent.; in the district of Zolkiew (1840), according to Seidl, out of 1,519 cases 196, *i.e.*, almost 13 per cent.; at Nagold, according to Schüz, nearly 10 per cent.; at Altdorf, according to Kapff, 10 out of 95 cases; at Herrenberg, according to Fricker, 1 out of 11; according to other physicians at Würtemberg, in the years 1836–37, out of 317 cases, 47; out of 312, 22; out of 266, 24, etc. Under the influence of specially harmful agencies, similar or still more unfavorable proportionate ratios are exceptionally encountered. Thus there died on the river Amazon, 1749–50, according to d'Alvea, 30,000 Indians; a similar mortality occurred in British North America, according to Meyer-Ahrens; at Madagascar, 5,000 cases died in one month in 1806; in the American army, according to Woodward, out of 21,676 cases of measles, over 2.5 per cent. perished, merely from the fever, without reckoning the numerous complications; the mortality was very considerable during the well-known epidemic of the Faröe Islands, also, according to Meyer-Ahrens, during that of Iceland (1846). There succumbed upon an Indian emigrant ship, according to Roux, out of 43 cases of measles, 11; in the restricted accommodations of the Children's Hospital at Stockholm, of 131 cases, 36 per cent. died; under unfavorable hospital conditions, according to Laveran, 40 died out of 125 soldiers who had contracted measles, but who were at the same time worn out by the campaign.

The causes of the malignancy of individual more or less extended epidemics of measles are, for the most part, unknown: they have been, in past times, unjustifiably sought only in the geographical position, character of the soil, special climatic influences, etc., and thus, according to Hirsch, the unsuitable therapeutic and dietetic methods of the day have been left entirely out of consideration. Even this, however, does not explain the differences of mortality which one and the same physician observes during different epidemics, or which have been reported from the same region or district at different times.

Thus Lippe, in middle Hungary, had in 1856 an epidemic so malignant throughout that over 50 per cent. of the cases died, mostly after a normal prodromal stage, through complications which occurred after the fifth day, while in 1863 only 8 per cent. died. The epidemic at Winschoten, beginning in May, 1865, occasioned, according to Tresling, a mortality of 4.83 per cent., while that of the middle of September, 1871, caused one of only 2.1 per cent. According to Karajan, the mortality of the epidemics of 1862, in lower Austria, which occurred during the presumably unfavorable cool months, reached only 2.29 per cent., while that which occurred in the summer months of 1863, in the same district, attained to 6.29 per cent. If from this summer epidemics should appear more fatal, yet in other places precisely the reverse has been the case. Thus, according to Voit, there died in the Children's Clinic at Würzburg, within thirty years, during the winter months, 12.7 per cent. of the measles cases; in spring, 11.5 per cent.; in summer, only 2.5 per cent.; in autumn, 0.4 per cent. According to Passow, however, of all the fatal cases of measles in Berlin, from 1863 to 1867, there took place in winter, 41.4 per cent.; in spring, 11.9 per cent.; in summer, 13.3 per cent.; in autumn, 33.4 per cent.; the autumn was, therefore, essentially more unfavorable than in Würzburg.

It is therefore clear that universally applicable rules cannot as yet be laid down, and even the rare exceptions of malignant epidemics must still await their explanation.

The mortality from measles varies at different places, as we learn especially from the compilations of Schiefferdecker. According to him there are due to measles out of 1,000 deaths in London, according to statistics extending over eleven years, 27.0; in Frankfort-on-the-Main (for a period of twelve years), 12.0; in Königsberg (for the same length of time), 9.2; in the Canton of Geneva (for thirteen years), 6.6; in Stuttgart (for fifteen years), 6.3; in Munich (for seven years), 5.8; in Berlin (for eighteen years), 3.8 cases. In some small places the difference may partly depend upon the presence or absence of severe epidemics of measles during the years concerned, but this cause does not at all explain the very noticeable difference between the two great cities, London and Berlin, in which it may be presumed that measles continually occur, and the cause must therefore be sought in local conditions, which either increase the mortality from measles in London, or that from other causes at Berlin: the former is probably the most important cause of the marked difference.

The influence of the mortality of measles upon the mortality of children generally, has been especially studied by Schiefferdecker.

Thus at Königsberg, according to the statistics of six years, among 1,000 deaths 4.2 were due to measles during the first year of life; 23.3 during the second year, 16.8 during the third, 17 during the fourth, 21.6 during the fifth year. Statistics of eighteen years give for Berlin, at the corresponding ages, 3.2, 13.7, 15.4, 17.3, 12.7;

of eleven years for London, 20.0, 100.9, 111.8, 96.5, 78.0. According to Gregory, there died of measles in England and Wales in 1838, 6,514; in 1839, 10,937; in 1840, 9,326 persons.

In hospitals and barracks, in a word, in too crowded localities, the prognosis of measles is often less favorable, partly owing to the deteriorated air prevailing in such places, partly from the possibility of other attacks of different infectious diseases, the contagious principles of which are often, so to speak, domiciled in badly arranged hospitals (separate buildings for special diseases exist only in very few places), and are more easily propagated amongst a closely packed population with its excessive intercourse than under ordinary circumstances.

Poor people who are usually badly nourished and often cachectic, scrofulous and anæmic, die to a greater extent than those in better circumstances. Lievin proved this by extensive estimates for Dantzic, while he was unable to show that a similar influence was exerted by a too dense population.

TREATMENT.

The most effective prophylactic measure is the isolation of those affected by measles. If this could be strictly carried out under existing circumstances, it would doubtless prevent the spread of epidemics, for the poison of measles is reproduced by infected organisms alone, and is communicated only by intercourse. Such isolation cannot, however, be reasonably demanded at present, and we must therefore give our minds to opposing, in the interest of individuals, the evils brought about by an essentially unimpeded spread of the contagion. To this end it is demanded, not unjustly from a purely medical stand-point, that the brothers and sisters of affected children should be forbidden access to all schools and institutions for a certain time, in order that the disease may not be transmitted by means of objects which have come in contact with the contagion, such as, especially, the clothes of the healthy members of the family. Such measures thoroughly enforced would certainly remove an important source of infection, thus guarding those disposed to the disease from an attack of it at a period of life when the prognosis is more unfavorable. Such a prohibition, however, is often enough evaded because it seriously interferes with the education of the children, nor can it be extended to the usual domestic intercourse, especially that between families; and thus ample opportunity for infection is always afforded. Nor is even the above measure of itself sufficient, for it does not prevent the possibility of infection from a person in the stage of incubation or even in the prodromal stage, if this has been overlooked. The closing of schools and similar institutions entirely upon the appearance of an epidemic would doubtless interfere materially with its extension, but would not permanently prevent its development; moreover, the measure would be superfluous in the usual mild epidemics, and at most only to be made use of when these were malignant. In private houses isolation is usually without effect, contagion occurring during the unrecognized prodromal stage; the measure is therefore superfluous. But when it is necessary to protect very young people, or those already ill with some other disease, from a secondary and oftentimes dangerous attack of measles, the physician must represent clearly to the friends the importance of isolation, and insist upon its being carried out with great strictness. It is generally considered unnecessary to seclude healthy children of more advanced age during a mild epidemic, but, as the prognosis is not absolutely favorable, the physician should not advise against such a measure. In children's hospitals, measles should in all cases be treated in separate wards, though in hospitals for adults this measure would be superfluous, as all the inmates of the ward would probably be no longer susceptible. Finally, care should be taken that the clothes and other effects of patients who have recovered be submitted to thorough disinfection before they come again into contact with healthy individuals, and this involves complete purification in an apparatus set apart for the purpose, by means of effective washing, and not through simple exposure to the air.

The greatest cleanliness and frequent change of the air in the sick-chambers of patients with measles,—where, if possible, a window should be continually open—benefit not only the patients themselves, but also those in their neighborhood who may perhaps be disposed to an attack, by preventing the infectious material from working in a too concentrated condition upon the latter. Even if this axiom cannot be positively proved in every individual instance, yet experience shows that an anomalous and complicated course of the disease, with its previously described evil consequences, is rather to be expected in badly ventilated and crowded rooms than a normal and mild course. The contagiousness is also probably diminished by frequent bathing of the patients. Other prophylactic measures and methods which have been employed are, as experience shows, of no use, or at least uncertain: the latter is especially true of inoculation, which, however, under suitable circumstances, might be empirically employed to procure further experience.

In the treatment of measles, as of the acute exanthems in general, the axiom must be borne in mind that the disease in its natural typical development cannot be interrupted, and leads to recovery, provided the fever and the local disturbances remain within their normal boundaries, and no dangerous complications intervene. The physician has therefore nothing more to do than to watch the course of the disease, to oppose injurious influences, and to place the patient under those circumstances in which interferences with the normal course are as far as possible obviated. All this is accomplished if the patient is enjoined to take to his bed, his diet suitably regulated, his thirst quenched with water, his chamber ventilated and kept at a temperature of about 63° to 67° Fahr., and somewhat darkened in his immediate neighborhood. During the preliminary stage, children with perhaps considerable fever at evening must be prevented from running about or going out of doors. In simple measles the normal development of the eruption upon the skin should be neither retarded nor promoted with useless zeal, *i.e.*, patients should neither be kept too cool nor foolishly heated with heavy bedclothes, warm drink, etc.; the former course is especially to be avoided, if fever is lacking, and the latter should certainly not be followed where the fever is severe. Simple paleness of the eruption, with slight fever and anæmia, demands no interference, at most some slight additional warmth; too marked a redness, usually accompanied by severe fever, may, on the other hand, need cool applications. The physician must prevent as far as possible any irritation of the mucous membranes affected, partly to get rid of annoying symptoms, partly to guard against more or less threatening dangers. If the eyes are congested, light should be excluded, the secretion frequently removed with lukewarm water, and, if necessary, cold applications employed. For severe coryza, water of an agreeable temperature or moderately warm vapor should be drawn into the nose. If the cough is violent, the air of the room should be of a moderate temperature and frequently changed, the patient of course being protected from any draught. If more is needed, and severe nervous irritation is present, the neck may be slightly protected with flannel, and mucilaginous, sweet, warm drinks administered, if thirst and heat permit; if marked though simple laryngitis is the cause of the cough, cold compresses about the neck are indicated. At times cold packings succeed in removing an immoderate tendency to cough. Narcotics should be employed for this purpose very carefully, in doses corresponding to the age of the child, and when a tormenting cough, preventing sleep, cannot be otherwise relieved. The cough may also be checked by an emetic given early, but care must be taken not to give too large a dose, especially if tartar emetic is employed, for fear of exciting a diarrhœa. Irritation of the gastric and intestinal mucous membranes is most easily prevented and treated by prescribing a suitable diet devoid of substances difficult of digestion, flatulent, fatty, or laxative. If constipation is present, which is rare, laxatives may be employed, but only the lightest forms (calomel excepted); usually simple injections are enough, while purgatives should only be employed after the expiration of the disease. Diarrhœa, which is often present and frequently severe, should be treated by cold compresses frequently renewed, or even by bladders of ice upon the abdomen; if less severe, by moist heat and by the limitation and still more careful selection of the food taken. Narcotics are only to be used in extreme cases; emulsions and astringents are preferable; revulsion to the skin by packings, with or without subsequent hip-baths, may succeed, and can at all events be tried if the application of a more intense cold to the abdomen is contra-indicated or refused. The usually slight angina accompanying measles, and the hyperæmia of the buccal cavity demand no special treatment beyond cleanliness of the parts.

The most important thing is the suppression of immoderate fever in the prodromal and especially in the eruptive stage. Even in the last century a few physicians employed cold for this purpose in the form of douches, baths, and spongings (Hahn, Currie), and Frölich and Thaer at the beginning of the present century drew up tables in regard to the warmth of the water to be used, which presupposed the thermometrical measurement of the temperature of the measles patients. At present cool baths, packings, and extensive cold compresses are the usual means employed; spongings are also admissible, though wiping must be omitted on account of its unavoidable irritation of the skin. The advantages of a judiciously administered cold-water treatment in measles are, that it usually affords to the patient more speedily and safely than any other antifebrile method a certain sense of comfort; that it is not apt to weaken or otherwise act unfavorably, and that it shortens convalescence by permitting the patient to expose himself to the fresh air sooner than under any other treatment (Mettenheimer). Moreover, it favors the cleanliness of the body, a thing of the greatest importance for the prevention of certain complications. While it is indispensable to combat, by means of cold, a high and dangerous fever, it is no less advantageous to moderate betimes a fever which tends repeatedly to exceed definite limitations. Compresses and packings should therefore be employed as soon as the temperature of the axillæ approaches or exceeds 102° Fahr., and baths once or twice daily as soon as it remains at 103° Fahr.; with higher temperatures, especially in young children, both these means should be united. The physician should proceed systematically, and not leave it to the relatives to determine the time and method of reducing the temperature, except where it is absolutely necessary; for the friends of the patient usually feel called upon to act only when severe nervous symptoms and the unmistakable signs of excessive heat are present. From the prudent application of cold one need not fear in the least any unfavorable influence upon the intensity and duration of the eruption upon the skin. The sensitive nervous system of children forbids, of course, the use of very cold baths, especially when the skin is very much overheated; here we should rather employ exclusively the gradually cooled baths of Ziemssen from about 90° to 77° Fahr. Cough and diarrhœa afford no contraindications; on the contrary, they are frequently quickly and decidedly improved, especially by packings; even the congestion of the skin, evidenced by the frequent sweating, is a very good means, according to Hofmann, of furthering the development of the eruption and also of hastening the expulsion of the contagious matter from the body. In general, cold water may be employed more boldly in the prodromal and eruptive than in the desquamative stage. If, however, cold water has been employed from the beginning, there is nothing to fear from its continuance; otherwise it is better to begin with lukewarm baths, or with somewhat cooler ones containing salt (artificial brine baths, according to Schwalbe), and proceed by slow degrees to cold washings and frictions, in order to harden the skin and not to increase the catarrhal symptoms. Moreover, the patient must under all circumstances, as long as fever is present, and especially with severe cough and objective signs of a bronchitis of the finer bronchial tubes, keep to his bed, and only leave his room in favorable weather, when the cough and other important symptoms of disease have essentially disappeared. Hauner says, and not without reason, "in no disease is careful nursing and attention so necessary as in measles, since with improper treatment and neglect the frequent and disastrous sequelæ will not fail to appear." "It would be well as a sanitary measure if the masses could be instructed in some suitable manner as to how they should treat their children when attacked by measles."

If the family refuse to allow these hydro-therapeutical measures, in the severe cases inunctions of lard or oil may be employed from two to four times daily, till the end of the eruptive stage. These, according to Schneemann, Walz (who out of 343 cases had but three fatal ones), Scoutetten, Mauthner, and Cornaz, alleviate the febrile symptoms and prevent sequelæ, and also exercise a favorable influence upon the bronchitis, etc. If the fever is very high, one may use in such cases quinine, which Binz warmly recommends for children, and which plays so important a rôle in the treatment of typhoid fever, but not in too small doses. I have often enough given children twenty grains a day, divided into two doses, without any evident result, certainly without injury. Smaller doses are of no value. Roncati and others, especially Italian physicians, recommend the daily administration of two drachms or so of sulphate of magnesia, until the end of the eruption, as a febrifuge and to prevent severe brain symptoms. Brown had the whole skin rubbed several times daily with diluted oil of turpentine, with the best results, especially as regards the bronchitis. These are harmless methods and require, in addition, merely the thorough ventilation of the sick-chamber.

A rational treatment during the normal course of measles is the best prophylaxis of complications and sequelæ. Deviations from the normal course of the temperature, and especially immoderate exacerbations of the fever, are usually the first manifestations of the development of complications and anomalies in the course of measles, or point at least more definitely than any moderate local symptoms to the danger which threatens the patient; a careful examination of the state of the temperature throughout, by means of the thermometer, is, therefore, the first condition of the proper treatment of measles. Not less to be regarded, however, are the abnormal local signs of disease: particularly severe nervous symptoms, usually the result of the increase of fever; thoracic symptoms with broncho-pneumonia; also intense diarrhœa with violent and extensive disease of the intestinal tract. Already in earlier decades a distinction was made from this practical stand-point between typhoid, inflammatory, and gastric measles, according to the special predominance in the course of the disease of nervous, thoracic, or abdominal symptoms.

Moderate deviation of the fever from the normal type requires only the usual treatment, with greater care; premature fever, violent in the prodromal and lasting strongly during the cruptive stage, calls for immediate and most energetic treatment by means of frequently renewed compresses and packings, or cool baths, in order to oppose at their very first appearance the graver anomalies and to calm the nervous system, the irritation of which can be manifested by delirium, sopor, jactation, and even convulsions; good results from this method as a rule are easily perceived. If this cannot be done, the milder anti-febrile measures above mentioned are to be employed. De Keghel employed with good results, as a revulsive in cerebral congestion, woollen stockings dipped in cold water. He employed the same means for feeble development of the exanthem, with severe brain symptoms and high fever, with the effect usually of causing perspiration and a normal eruption. For the violent and long-continued convulsions of a child of four months sick with measles, Pinkham gave chloral hydrate in two-grain doses every twenty to sixty minutes, with the best results, a proof that even convulsions, which as here cause the disappearance of the exanthem, admit of treatment; initial convulsions in measles are usually not dangerous, if not too intense or frequently repeated, and require, therefore, no special treatment. If after the longer or shorter duration of severe brain symptoms the energy of the heart begins to sink, an unfavorable result may often be averted, or deferred at least, by the immediate administration in large doses of stimulants (wine, benzoic acid, camphor; if the surface is cold, warm baths and cool douches upon the head, irritants of the skin, etc.).

Severe capillary bronchitis, the precursory stage of broncho-pneumonia, may at first be treated by one or more emetics, the preparations of antimony being avoided on account of their action upon the intestine. Emetics, however, are usually only of service when the larger bronchi are crowded with mucus; the violent affection of the smaller and smallest bronchi must be met by calling forth prolonged and deep inspirations, and by revulsion to the skin. As regards the genesis of broncho-pneumonia, Bartels has especially alluded to the great importance of thorough ventilation of the sick-chamber, for he has shown that this complication appears preferably in the small, low, tightly closed, foul-smelling, crowded dwelling and sleeping chambers of the poorer population. The deleterious influence of such dwelling-places upon the bronchitis of measles he ascribes largely to the circumstance that they contain an excess of carbonic acid, which prevents the elimination of this element from the blood, and also causes the energy of the respiratory movements to become so weakened that collapse of the lungs and pneumonia must almost necessarily follow. The resulting mortality of the epidemics which he observed lasted until the population became frightened at the high death-rate, and then at last followed the directions of physicians in regard to ventilating the sick-chambers. In summer. patients may be carried with certain precautions into the open air. It is just as important to pay heed to the skin as to the organs of respiration, if we wish to avert or cure the complicating pneumonia of measles or to cure the disease itself. Though the old sudorific treatment, the purpose of which was to occasion a more marked development of the exanthem, under the idea of thus relieving the internal organs, has been given up by physicians in the treatment of measles, nevertheless prejudices are still entertained by many against the use of baths, even warm, on account of the supposed possibility of their exerting an unfavorable influence upon the cough and catarrh of the air-passages in general. It is to be hoped that the favorable results of hydrotherapeutics may overcome these prejudices, and that ventilation and cleanliness may, in future epidemics, gradually cause pneumonia and the other dangerous complications of measles, and with them their mortality, to sink to an unavoidable minimum. The most advantageous method of treating the high fever of the broncho-pneumonia of measles, according to Bartels, Ziemssen and others, is the following: several thicknesses of cloths wrung out in cold water are laid upon a piece of flannel of sufficient width to protect the bed-clothes from becoming wet; the naked patient is then placed upon these and enveloped in them. Lively kicking and screaming ensue, giving depth and force to the previously superficial inspiration; by degrees the children become more quiet, and soon fall asleep. The cold wrappings are to be renewed every half hour, or less, until the temperature, pulse, and frequency of respiration are markedly diminished, which is usually the case in a couple of hours. The wrappings are then removed, the skin dried, the children clothed in clean, warmed garments, moderately covered up, and left to lie until a new exacerbation of the fever and of the dyspnœa, or of the pains in the chest, renders necessary the repetition of the wrappings. This point is to be especially impressed upon the attendants. It is only in exceptional cases that the wrappings must be continued uninterruptedly for several days and nights, or that we are obliged to resort to the use of baths and cold douches upon the head and back. This mode of treatment may have to be repeated daily for weeks, according to the duration of the pneumonia. Wrappings are usually preferable to the use of more energetic antifebrile methods, since the cooling of the body is more gradual, and therefore irregularities in the circulation and distribution of the blood are probably better guarded against. They obviate also any necessity for the use of medicine. The cough is generally relieved by the easier expectoration of the mucus, and the pains in the chest and the febrile symptoms, as manifested in the peripheral organs, are usually diminished in proportion to the effect produced. It is not necessary, according to my experience, to employ other febrifuges, such as digitalis, or quinine in large doses; I strongly recommend, however, the use of stimulants, especially wine. Now and then, when the bronchi are filled with mucus, an emetic may be of use (apomorphine, ipecacuanha). Trousseau especially recommended, when the chest symptoms were severe, as a revulsive to the skin, urtication, *i.e.*, whipping the whole skin with nettles, while Brown advised inunctions with oil of turpentine. A diet as nourishing as possible under the circumstances will essentially aid the therapeutical measures, which, if a certain temperature is kept up, under the control of the thermometer, can hardly exercise any injurious effect upon the exanthem, especially in the beginning.

Carefully wrapped packings are especially to be recommended, if, apart from the antifebrile removal of heat, a revulsion to the skin in a marked degree appears desirable. This is the case with diarrhœa, which is often quite violent, and does not always exercise a favorable influence upon the chest symptoms. With remission of the fever, and after continued diarrhœa, a moist abdominal bandage generally suffices; this acts like a moist compress of the same temperature as the body, and causes by degrees the relaxation of the vessels of the skin, and thus a favorable determination from the intestines; it must be allowed to remain until nearly dry. With dysenteric, muco-sanguineous discharges, it is at times advisable, according to Mosler, to wash out the large intestine with a large amount of water; so also to employ ice clysters liberally for severe hemorrhages. On the other hand, moderate injections of cold water, repeated several times daily, are especially advisable if the upper intestinal tract is alone concerned. A moderate amount of fresh water is the best drink to appease the tormenting thirst. With adults and older children appropriate doses of opium in a mucilaginous vehicle are of benefit; with smaller children these should be avoided or only very cautiously employed. Astringents, as possessing no toxic action, are better suited to these cases, though their taste often causes a sufficient dose to be refused.

The treatment of the other local affections is modified by the presence of measles only in so far as the avoidance of debilitating influences of all kinds, especially losses of blood, is concerned; in the case of gangrenous complications and affections of the intestinal canal, mercurials and strong purgatives should be avoided to the utmost. The following affections alone require special mention.

Slight epistaxis in the prodromal stage demands no special treatment, is even often of benefit; profuse epistaxis requires the head to be raised, the arm of the side affected to be held up, a cooled head, antifebrile remedies, local cold applications, astringent injections, compression of the nasal apertures if the bleeding is from the front portions, and ergot in case of need. These will generally suffice, and render unnecessary the troublesome application of mechanical contrivances. Trousseau especially recommends injections of water as warm as possible. Ozæna is to be treated as usual, that is, by frequent cleansing and the use of disinfecting and deodorizing injections.

Marked affection of the eyes requires the energetic application of cold and frequent cleaning of the parts, and at a later stage somewhat astringent compresses and eye-waters.

Light cases of aural affections often recover of themselves, but may, according to Wendt, demand at times a special treatment. The object of this is the alleviation of existing pain (local bleedings, frequent filling of the auditory passage with warm water, poultices); restoration of the normal atmospheric pressure in the middle ear (forcing the air in by the so-called Politzer's method); removal of the secretion from the nose and pharynx (nasal douches, not too often within twenty-four hours; gargles), and also from the external auditory canal, when perforation of the membrana tympani is present (injections).

The more intense laryngeal affections demand special consideration. Cold wrappings or Priessnitz's compresses over the region of the larynx answer the purpose at first, especially with otherwise high fever. If pseudo-croup appears (hoarse cough, whistling inspiration, protracted expiration), diaphoretics are needed, and the neck is to be sponged lightly with pretty hot water, but not so as to burn the skin; the resulting determination of blood to the surface occasions the remission of the threatening symptoms. Should these, however, continue, emetics must be given until they act; if no benefit ensues, and symptoms appear of œdema of the glottis, warm compresses should be tried before having recourse to the last means of saving life, tracheotomy. The same treatment is adapted to true croup. In paroxysms like those of whooping-cough, narcotics may be of service, if the age of the child renders them admissible. Where the laryngeal trouble is subacute and tends to become chronic, blisters may be applied to the neck; the surest result is guaranteed by the inhalation of atomized fluids, or by cauterization for ulcerations, and here with adults the laryngeal mirror should be employed. West was in the habit, in croup, of cauterizing with a little sponge fastened to a piece of whalebone and dipped in a strong solution of nitrate of silver. Leeches should not be employed for fear of too great loss of blood from subsequent bleeding, and consequent weakness. The diet should be a very nutritious one to prevent chronic respiratory troubles and tuberculous affections.

Gangrene is to be prevented by careful cleanliness of all parts of the body, especially of those which manifest slight acute or chronic affections, excoriations or wounds, and in hospitals by isolation from patients already attacked by it. If it is already present it needs very energetic treatment. If we wish to restrict the gangrenous ulcer to a small tract, the parts attacked must be cauterized with concentrated hydrochloric acid and other caustics, even with the actual cautery, and then bandaged with disinfectants. Bathing must be zealously kept up, and the diet made as strengthening as possible.

Complicating diphtheria of the pharynx is to be treated in the usual manner, with frequent purifications of the mouth, and by gargles containing suitable disinfectants (chloric, acetic, or carbolic acids), or by antiphlogistics (cold bandages around the neck, ice in the mouth), but not by cauterizations. If in this way the progress of the affection to the trachea cannot be prevented, there is here nevertheless a much better prospect of saving the patient's life by tracheotomy than in the diphtheria of scarlet fever; under Abelin, at least two out of six who had been thus operated upon, recovered.

In the treatment of the diphtheria of other mucous membranes, the same therapeutical principles are to be observed.

RUBEOLA.

(ROETHELN, GERMAN MEASLES.)

Writings which do not regard rubeola under the acceptation of the present day, or do not decidedly advocate this acceptation: *Orlow*, De rub. et morbill. discrim. Progr. Königsb., 1758.—*Willan*, Hautkrk. übers. v. *Friese* 1798–1816.—*Heim*, Hufel. Journ. Bd. 34. p. 76. 1812.—*Wolf*, Hufel. Journ. 1812. Bd. 34. 4 St. p. 69.—*Henke*, Hdb. d. Kndrkkh. 1818.—*Meier*, Badische Ann. 1828. III. 2. Heft. p. 129.—*Jahn*, Anal. üb. Kinderkkh. 4. Heft. p. 150. 1835.—*Fuchs*, D. kkhft. Veränd. d. Haut. p. 1063. 1841.—*Schönlein's* Path. 5. Aufl. II. p. 272. 1841.—*Meissner*, Kndrkkh. 3. Aufl. II. p. 582. 1844.—*Canstatt*, Hdb. II. p. 278. 1847.—*Kronenberg*, Journ. f. Kndrkkh. 4 p. 244.—*Simon*, Hautkrk. 1848.—*Naumann*, Pathogenie 1841. p. 292.—*v. d. Busch*, Oppenh. Zeitschr. p. 289. 1851.—*Paasch*, Journ. f. Kind. Bd. 24. p. 74. 1855.—*Bednar*, Lehrb. d. Kinderk. 1856.—*Wunderlich*, Path. 2. Aufl. 1856.—*Küttner*, Journ. f. Kind. Bd. 30. p. 180. 1858.—*Gelmo*, Jahrb. f. Kindhk. I. Ser. I. p. 152. 1858.—*Lebert*, Hdb. d. pr. Med. 1859.—*Th. O. Heusinger*, Diss. inaug. 1860.—*Gintrac*, Canst. Jahresb. 1858. Journ. de Bord. p. 545. 1862.—*Cless*, Würtemb. Corr. 1862. Nr. 16. Bd. 32.—Bericht etc. Ibid. Nr. 20.—*Niemeyer*, Path. 5. Aufl. 1863.—*Kunze*, Compend. 2. Aufl. 1865.—*Köstlin*, Arch. d. V. f. wiss. Heilk. II. p. 338. 1866.—*Henoch*, Beitr. z. Kindhk. 1868.—*West-Henoch*, Kindkkh. 5. Aufl. p. 476. 1872.—*Foss*, Edinb. Journ. p. 280. 1872.—*Seitz*, Bay. Intell. 1873. Nr. 51.

The following advocate a specific rubeola: *Wagner*, Heck. Ann. 1829. XIII. p. 420. Hufel. Journ. 1834. 79. Bd. 2. St. p. 55.—*Collin*, Hygiea 12, p. 347. Schm. Jb. Bd. 76. p. 66. 1852.—*Salzmann*, Würt. Corr. 1862. p. 153.—*Faber*, Ibid. 1861. p. 326.—*Thierfelder*, Greifsw. med. Beitr. Bd. II. Ber. p. 14 1864.—*Hennig*, Kindkk. 3. Aufl. 1864.—*de Man*, Arch. f. d. holl. Beitr. III. p. 1. 1864.—Prag. Vtljschr. Bd. 74. p. 44.—*Danis*, Diss. Strassb. 1864.—*Trousseau*, Med. Klin. p. 158. 1866.—*Veale*, Edinb. M. Journ. 1866. p. 404.—*Arnold*, Bay. Intell. 1867. Nr. 40.—*Wunderlich*, Eigenw. 1. Aufl. 1868. p. 320.—*Thomas*, Jahrb. f. Kndrhk. II. p. 233. 1869.—V. p. 345. 1872.—*Steiner*, Arch. f. Derm. I. p. 237. 1869.—*Mettenheimer*, Journ. f. Kind. Bd. 53. p. 273. 1869.—*Squire*, Brit. med. Journ. Jan. 29. 1870.—*Emminghaus*, Jahrb. f. Kind. IV. p. 47. 1871.—*Gerhardt*, Lehrb. d. Kind.

1871. p. 74.—*Vogel*, Lehrb. d. Kind. 5. Aufl. p. 416.—*Dunlop*, Lancet. II. p. 464. 1871.—*Fleischmann*, Wien. med. Woch. 1871. Nr. 29–31.—*Kunze*, Lehrb. 2. Aufl. 1873.—*Liveing*, Lancet. March 14, 1874. I. 11. p. 360.

HISTORICAL NOTICE.

THERE are few diseases in regard to which opinions vary so much as about rubeola, or rather about that which is designated as rubeola by various authors. The work of separation and discrimination, which has by degrees given us a definite conception of the other acute exanthems, has by no means reached its final result as regards this. Until very recently it has seemed to many so difficult to bring order out of the chaos of rubeola, that, in their writings, they have either passed it over in almost complete silence, or have preferred to adduce, almost without criticism, an orderly array of individual opinions.

The name "rubeola" was brought into use by German physicians about the middle of the last century for an acute exanthem, which, according to the concurrent observations and experiences of all, could belong to no one of the acute contagious or non-contagious cutaneous eruptions, though closely resembling measles and scarlet fever. Somewhat earlier still, we find in English and French writings the description of a similar exanthem, to which was applied the name "roseola,"—the term rubeola being used by them to signify measles. The history of medicine throws but little light upon the origin of rubeola. Some affirm that it was known to Arabian physicians under the name "Hhamikah;" others, that it was first recognized about the middle of the last century, from which time the more accurate descriptions date (Orlow, 1758). Whether the benignant "Rossalia epidemics" of earlier centuries were rubeola or not, cannot be ascertained, owing to the lack of accurate descriptions.

Partly owing to the natural confounding of the rubeola of the Germans with the "rougeole" of the French, partly owing to the vagueness of type of rubeola and roseola, partly on account of the lack of opportunities for observations of true rubeola in hospitals, there now arose that inconceivable mystification in regard to this disease which has not even yet reached its end. Many, under a more or less clear impression of the existence of rubeola, considered themselves justified in regarding as true rubeola an acute, red, somewhat peculiar eruption of short duration, and in doubting the genuineness of the rubeola of others, the result of which was, that various mutually dissimilar forms of disease were described under this name, and the true rubeola was nearly lost sight of in the confusion. Thus Göden in 1822 confounded rubeola and scarlet fever, and taught that the former was a protection against the latter; that both, however, only appeared in combination. Jahn, in 1835, was convinced that rubeola did not exist, and that the cases observed were for the most part cases of scarlet fever. Even as late as the middle of this century, rubeola, in accordance with this idea, was for the most part regarded as an anomalous scarlet fever, and the celebrated Heim even states that it is more dangerous than the latter (according to Formey there died between 1784–94, in Berlin, 457 (!) from rubeola, 172 from scarlet fever, 53 from measles), while others declared it a variety of measles. The natural consequence was the entire rejection, in many places, of the term rubeola and the distribution of these cases between measles and scarlet fever (Naumann); while others, regarding the peculiarities of rubeola as too well marked for this, preferred (v. d. Busch) to ascribe to it "a certain individuality," basing such an ascription especially upon the fact that epidemics of it could appear distinct from those of measles and of scarlet fever. They distinguished, therefore, in accordance with the varying appearances of the eruption, the rubeola of measles and that of scarlet fever (rubeolæ morbillosæ et scarlatinosæ), a division which has endured even till the present time (Niemeyer). It is extremely probable that much of that which has been described under this name was nothing more than measles or scarlet fever, and that the diagnosis of rubeola was often made merely to conceal the embarrassment of the diagnostician; as to some reports, however, this view is untenable. Others imagined, with Hildenbrand and Schönlein, that they had unvailed the mystery of rubeola when they assumed that this was no indefinite disease, but a hermaphroditical form of measles and scarlet fever, or at least a disease lying midway between the two, the supposition being that an infecting agent of a special nature was generated outside the organism during the simultaneous prevalence of epidemics of measles and scarlet fever; while at another time a simultaneous infection with both contagions was regarded as the cause of the special affection of the individuals. Schönlein explained the variation in the eruption during different epidemics by an antagonism in the symptoms of the skin and of the mucous membrane, the nature of which is manifested by the fact that when the symptoms of the mucous membrane resemble scarlet fever, the exanthem resembles measles, and *vice versâ*. This conception is still retained by some. Simon declared rubeola as partly anomalous scarlet fever, partly confluent measles. According to Canstatt, the conception of rubeola is rather vaguely limited, and under it may be comprised every red, blotchy exanthem, in regard to which the consideration of the general or mucous-membrane symptoms gives rise to doubt as to whether it should be looked upon as scarlet fever, measles, erythema, or urticaria. He regards it as an erythema, or an urticaria, or a "hybrid form of skin disease, which portrays locally the reflex action of very various genetically different conditions," and distinguishes, accordingly, a roseola scarlatinosa, morbillosa, typhosa, variolosa, vaccinica, cholerica, rheumatica or arthritica, æstiva, autumnalis, gastrica, etc., and considers it unjustifiable to assume a peculiar rubeolous morbific process which cannot be included in this category. Hebra objected to several of these forms of roseola,—which, it should be stated, had already been set forth by Willan and his followers—on the ground that unbiassed observation teaches that such roseolæ represent either light cases of measles, with or without moderate catarrh, or cases of an imperfectly developed exanthem of scarlet fever of moderate intensity, or even, finally, cases of urticaria. He believed it, therefore, entirely superfluous to describe an individual exanthematic eruption under the name of roseola or rubeola (Rötheln). West expresses himself similarly; for, according to him, when rubeola is neither scarlet fever nor measles, it may be regarded as nothing more than a simple case of roseola, in which nothing specific can be detected.

Gelmo, in accordance with Hebra's views, and in spite of an apparently favorable opportunity for observations outside the hospitals of a great city, divides the rubeolous exanthems, and refers them partly to an anomalous form of scarlet fever,—the scarlatina variegata of Hebra—partly to confluent measles, and partly to a non-specific roseola, this last occurring only in sucklings, and consequently desires that the whole conception of rubeola should be dropped, since the different cases comprised under it can be easily included in these three well-characterized species.

Later observers, however, like the earlier ones, are continually manifesting their discontent with these attempts at explanation on the part of greater and lesser authorities, and adducing from their own experiences a number of cases, resembling each other in character, which cannot, like those of their predecessors, be brought under any of the proposed schemes, but force them to assume an independent rubeola. While, however, the minority of these content themselves with asserting the individuality of rubeola and deny its contagious nature, the majority, on the ground of the observation of more or less extended epidemics, allege its decided contagiousness, and declare it a specific acute exanthem, perfectly comparable to measles and scarlet fever, the diseases with which it was formerly usually confounded. The number of the advocates of the distinctness and contagiousness of rubeola has of late markedly increased, and there is a general effort to establish symptoms by which it may be as perfectly as possible characterized and permanently distinguished from non-specific forms of roseola, as well as from measles and scarlet fever with a somewhat anomalous exanthem. Among the older adherents of an independent and specific rubeola, I mention especially Wagner and Trousseau; Collin, Tripe, and Meissner have not been quite clear in their writings, nor uninfluenced by the indefinite opinions of their contemporaries; but, since the beginning of 1860, the specific nature of rubeola has been defended decidedly and with ever-increasing success by Thierfelder, de Man, Hennig, Dnais, Veale, Mettenheimer, Lindwurm and Arnold, A. Vogel, Wunderlich, Dunlop, Squire, Gerhardt, Emminghaus, Küster, and myself.

ETIOLOGY.

The assumption of a specific rubeola is based chiefly upon the fact that at certain times epidemics appear, the distinct cases of which show by definite symptoms an unmistakable mutual relationship, and conversely also a difference from scarlet fever and measles upon the further fact, that, as in the case of other infectious diseases, one attack of this affection affords almost perfect protection against a second similar one, but none against an attack of measles or scarlet fever, just as these afford not the slightest protection against rubeola. It must certainly be granted that second attacks of measles and scarlet fever may occur; trustworthy cases of this are, however, exceedingly rare, so that the possibility is suggested that the occasional assertion of the contrary rests upon the confounding of measles and scarlet fever with their congener, rubeola. In favor of this supposition is the circumstance that those who regard second attacks of measles and scarlet fever as frequent, are exclusively such as do not accord to rubeola a specific character. A correct judgment in regard to sporadic cases is far more difficult than when epidemics of rubeola are present, since in a single case the extension by contagion cannot be taken into account, and we are limited solely to the consideration of the products of the disease. Certainty in such cases can only be attained by establishing accurately all individual peculiarities, and even then perfect conviction cannot always be secured. The fact must, however, be here borne in mind that very various causes can educe extremely similar forms of the affection of the skin. All other rubeolous exanthems, in the production of which the agency of a specific cause (measles, scarlet fever, variola, varicella, typhus, dengue, etc.) can be excluded, and which usually also in other respects afford sufficiently frequent differences from specific rubeola, are not to be classed under infectious, but under skin diseases. A slight reservation must here be made in regard to the infrequent, and therefore too little studied forms of roseola.

We are now prepared to confine our attention solely to the consideration of the contagious and essentially epidemic, and therefore specific and infectious exanthem, to which the name "rubeola" can alone be properly applied.

Starting with the incontrovertible fact that anomalous eruptions of measles and scarlet fever occur, the imperfectly characteristic nature of which is due solely to individual idiosyncrasies (which is proved by the fact they generate again, by contagion, cases which are perfectly normal), we must also not expect of epidemic cases of rubeola, connected by contagion, and therefore proved to belong together, that their exanthem should be in all cases of a precisely similar type. Different diseases of the skin may be due indisputably to one and the same cause, *e.g.*, syphilis, or even to one which is not infectious, *e.g.*, balsam of copaiva! A too minute and restricted consideration of the conditions of the skin is clearly responsible for the confusion which has thus far prevailed in regard to the nature of rubeola. The more liberal the opinions were in regard to the admissibility of the diagnosis of measles and scarlet fever, the more the conception of rubeola became restricted, and the more easy it was to attribute this to the indistinct picture of a symptomatic roseola. Without neglecting, then, the alterations of the skin, or undervaluing their diagnostic significance, it is yet considered at present impracticable to base the diagnosis of rubeola solely upon them, and the necessity is recognized of comprehensively considering also other important conditions.

Before the individuality of rubeola was properly recognized a distinction was often made between a form resembling measles and one similar to scarlet fever,—rubeolæ morbillosæ et scarlatinosæ. According to my observations the exanthem of rubeola possesses a similarity to that of measles only, not the slightest to that of a normal scarlet fever. I am willing to admit the possibility of the existence of an equally important specific affection with a scarlatinous condition of the skin, although with all watchfulness I have as yet never met with such a form. Older observations, which allude to such, have reference perhaps to nothing more than light cases of scarlet fever; at all events, nothing more accurate can now be advanced. Until, however, new and exact observations have furnished indisputable evidence that a scarlatinous form of rubeola exists, and that cases of it originate by contagion from the usual rubeola exanthem resembling measles, and *vice versâ*, I consider it necessary to carefully discriminate between rubeola proper and casual scarlatinous exanthems, possibly also such as indicate a specific origin.

Rubeola is especially a disease of childhood, attacking indiscriminately boys and girls, older and younger children, down to sucklings. Adults up to about forty years are not infrequently affected; young women are apparently rather more often attacked than men of the same age or older persons. After the fortieth year the susceptibility is nearly lost, and we may consider it as essentially weakened at puberty, and as steadily diminishing subsequently. Seitz reports a case in the person of a woman aged seventy-three.

I have never yet observed a second attack of rubeola in the same person; if it occurs, it is certainly as rare as a second attack of measles, etc. The opposite statement with regard to measles is supported only by the confounding of the same with rubeola. Emminghaus saw in one case a relapse of rubeola one day after the termination of the first eruption, in two other cases after fourteen days; in each case the previous eruption was much weaker than the succeeding one. Lindwurm also appears to have observed relapses. Köstlin, who describes accurately the peculiarities of rubeola, and yet regards it as a light form of measles, saw in the same epidemic several children attacked for the second time.

The contagiousness of rubeola is considerable, though somewhat less than that of measles. Thus at times a few children of a family in which rubeola is present may escape. It attacks the healthy as well as the sick. The contagious principle itself is still entirely unknown; the vehicles by which it is conveyed are probably the same as in the case of measles.

Epidemics of rubeola have not yet been sufficiently studied, doubtless from the mildness of the symptoms or because they have been regarded as light cases of measles. It is not known whether, as in the case of measles, a rotation of several years exists, though this appears to be the case. For example, epidemics occurred at Leipzig in the spring of 1868, and at the same season in 1872; in the interval only sporadic cases were met with, from which we may perhaps infer that rubeola may be, like scarlet fever, endemic in thickly settled districts.

I judge from experience that epidemics of rubeola are about as frequent as those of measles, as far as may be decided upon the ground of general estimation; for while one hears everywhere of the disease, only very few patients are registered. I have had the greatest difficulty in making any observations. Necessary relations between epidemics of rubeola and those of other infectious diseases do not exist. Our last epidemic at Leipzig in particular can in no way be considered as having been the result of the contagion of measles.

Pathology.

The normally developed eruption of rubeola consists of numerous discrete hyperæmic blotches from the size of a pin's head to at the utmost that of a bean, with at times a tolerably distinct outline or again a less colored and somewhat faded border, and usually rising slightly above the level of the skin, so that this feels a little rough. The eruption is due to capillary hyperæmia of the papillary body and of the uppermost layers of the corium; this can give rise to slight inflammation and exudation between the uppermost stratum of the corium and the epidermis, but it occurs only exceptionally in a few cases, and then only on single parts of the body, and involving only a minority of the spots. The spots are generally round or oval, and not indented, though at times moderately so, and then they are connected here and there with each other by delicate processes. Nearly all parts of the body may be attacked; but especially the face, which as a rule shows the eruption sufficiently well to distinguish rubeola from scarlet fever. About the mouth the spots are usually well defined. Their color can in a few cases and at the acme of the development of the eruption be quite bright, though not equalling the dusky red of a severe scarlet fever, nor the peculiar bluish red tint of intense measles. It is usually only a pale rose red, but suffices, however, for the most part to bring out the spots in marked contrast with the normal skin.

It is everywhere of about the same depth, the color even of the face not exceeding that of the other parts. On isolated portions of the skin, of greater or less extent, the spots do at times show more clearly than elsewhere. These variations of intensity occur, however, only when the exanthem is as a whole but moderately red, the slight partial excess being due to accidental collateral circumstances. I have never seen them where the eruption was strongly colored.

Apart from the color of the exanthem the characteristic most striking to the eye is the size of the separate spots. This is not always the same on all parts of the body in each individual case. In this connection three types may generally be distinguished: one with large spots, one usually with spots of medium size, and one with small spots. The first is rather rare. The spots are here proportionately sparse, but can attain the size of two-fifths of an inch square; they are never round, but angular and indented, and of the most various irregular forms. Though they determine the type of the eruption, yet they are never present alone, but are always mixed in the most varying manner with the smaller, and especially the smallest spots, which are wont to mass themselves noticeably around the larger spots, and in some measure to form groups with them. In the type with small spots the single spots are more crowded, and at more regular intervals; they are about the size of lentils, though many are smaller, only a few larger. The exanthem, therefore, resembles in some measure scarlet fever, the spots of which are, however, usually much smaller and more closely arranged. In the common type the single spots are least crowded, and are usually of the same size, namely, that of a lentil, though smaller and larger ones also exist. The type is chiefly determined by the moderate degree of crowding, and the non-preponderance either of the larger or of the very small spots. Transitions, of course, exist. Even on those parts of the body where the spots are most abundant, these are only moderately crowded together. They occur most profusely, and relatively most densely, though rarely confluent, upon the face, especially on the forehead, cheeks, chin, and produce here the appearance of a light œdema, particularly if any swelling of the lymph glands is added. They often lie quite closely crowded together on the neck and trunk, especially on the neck; more often, however, they are no more abundant than on the thighs and upper parts of the arms. Then, too, the posterior aspect of the thigh and the gluteal region are also not infrequently profusely covered with spots, the result of keeping warm in bed. The scalp is always attacked, and often severely. The forearms, hands, lower parts of the legs, and the feet, are generally less markedly affected, not only as regards the number and closeness of the spots to each other, but also as regards their size and color. Here the rather pale spots often occur only singly, though they are never wholly absent. On the palms and soles they are usually harder to find, since on account of the greater thickness of the epidermis they are more indistinct here than elsewhere; but careful examination will always detect them even here, if the exanthem is elsewhere tolerably profuse.

The rubeolous spots are generally discrete, without special tendency to become confluent. When this in rare cases does occur, it is not general, but most marked in the face (Mettenheimer), or upon the extremities. In the first epidemic which I observed, the spots upon these last were frequently so closely crowded together, and were so bound together by slender processes that the legs in particular looked as if they had been sprinkled. In the second epidemic, with fewer observations, the spots on these parts were more discrete, and the same was the rule also upon the palms and the soles. The duration of the spots was often scarcely two, but sometimes four days. In correspondence with this the duration of the period of their most marked development was only very short; a half-day was perhaps the longest, while it often lasted only a few hours. After the disappearance of the spots a very delicate and fleeting brownish or yellowish pigmentation often remained, or at times none at all, even when the color of the eruption had been marked. Desquamation was entirely absent in most cases; in a few there were traces of it referable rather to the dryness of the skin, with consequent exfoliation, than to the exanthem, the slight intensity and duration of which thay explain the absence of this symptom; it is never lamellar, as in scarlet fever.

In rare cases the exanthem undergoes a further development on certain portions of the body, especially on the back; this consists in the formation, upon the hyperæmic spots, of a varying number of vesicles resembling miliaria (rubeolæ vesiculosæ); they are nearly always of small size and often only rudimentary, and very probably owe their origin to external conditions, and chiefly to the influence of a high temperature of the air surrounding them. In such cases there of course occurs later a correspondingly slight branny exfoliation. Dunlop thinks that he has seen petechiæ at times; I have never found this condition.

The eruption was itchy in a few cases, as often occurs in sensitive children in the case of any other eruption. The symptom of itching is very frequently alluded to in the older descriptions; among the more recent it is especially mentioned by Vogel. An eruption like urticaria, such as sometimes occurs on a few parts of the skin, with an intense development of measles, I have never encountered in connection with the far more moderate redness of rubeola, not even with such eruptions as itched. Nor have I, any more than all later observers, been able to perceive any special odor of rubeola. It is also chiefly on account of the similar odor that Heim regards it as a variety of scarlet fever.

It cannot be overlooked that the exanthem of rubeola possesses a great similarity to that of measles, and this circumstance alone is perhaps the reason that even skilful investigators have regarded rubeola as a light form of measles. But a more attentive and repeated observation of the same soon affords, in every case, means to distinguish from each other, thoroughly and at once, the far larger majority of cases of the two diseases. These distinctive features pertain chiefly to the size, form, and color of the single spots. The size of the spots of rubeola is decidedly less, their form more round; they are not so angular and indented, nor so often provided with processes; owing to the very slight swelling, they seem paler and more as if they had been sprinkled over the surface. If the characteristics concerned were always and everywhere so sharply pronounced as in the majority of cases, no one certainly would ever confound measles with rubeola. But it is not so. Measles are not always perfectly typically developed, and in an exceedingly small minority of cases of rubeola the spots are large, indented, and bright colored, as in measles. These require, therefore, for the establishment of a diagnosis the consideration also of other circumstances ; since, however, these latter are not so patent as the exanthem, they are frequently neglected, whence the great confusion in the doctrine of rubeola.

Course of the Disease and Symptomatology.

As to the course of the disease, the observation of children in the incubative stage of rubeola has never shown any disturbance of the health with increase of temperature or other appearances of disease.

For the estimation of the duration of this stage of latency, I have only statistics of the interval between the beginning of the disease of the infecting, and that of the infected child,—data from which the actual duration of the period of incubation cannot be reckoned, since the act of infection, which is unrecognizable, need by no means coincide with the commencement of the disease in the first child. Noticeably often, however, in such cases, the second attack follows in from two and a half to three weeks, and it is therefore probable that this is about the duration of the incubative stage. In favor of a definite duration of this period, varying only within narrow limits, is the circumstance that members of one family, after a presumably simultaneous infection, also fall ill at exactly the same time.

The most important symptoms, after those of the skin, are afforded by the mucous membranes of the air-passages, and of the buccal and pharyngeal cavities. The former are almost always in the condition of catarrh, less intense than with measles, yet so that coughing and sneezing are rarely absent, especially in the beginning of the disease ; at times slight hoarseness occurs. Towards the end of the attack these symptoms of irritation diminish by degrees, with increase of the secretion. Congestion of the conjunctival vessels, burning pain in the eyes, and some photophobia are likewise frequently present. A somewhat congested condition of the mucous membrane of the palate is never absent. If this also appears in some measure confined about single foci, instead of being equally distributed over the palate, it is yet far removed from affording a so typically spotted picture as the skin offers. By the term mucous-membrane exanthem, as this condition is frequently called, we must not understand anything perfectly analogous to the eruption upon the skin. Upon the skin there is no sign of hyperæmia between the individual spots; the mucous membrane, however, is more or less affected as a whole, and the parts of it which perchance are normal by no means preponderate over the reddened portions, which, at least among the most numerous patients—the children—are distributed most irregularly in streaks and spots. In the case of an adult one would be more apt to find upon a universally injected mucous membrane single spots of a deeper red. The pharyngeal mucous membrane is usually somewhat injected, and at times even the tonsils are moderately swollen, in which case a painful difficulty in swallowing would also probably exist. The tongue constantly shows a white coating, pierced especially at the tip by separate, swollen, papillæ-like red knobs.

In conjunction with the affection of the mucous membrane of the mouth and pharynx, we find, though not invariably, according to the observations of different authors (Thierfelder, Mettenheimer), the lymphatic glands of the neck and nape generally moderately but often severely swollen, so that they can even be the cause of moderate œdema of the corresponding regions. Slight swellings of the lymphatic glands are also frequently observed on other parts of the body, although, as far as my observations are concerned, they are by no means always present. Chronic swellings of the lymphatic glands are of course very frequently to be found on children with rubeola. According to Thierfelder, swelling of the subauricular and superior jugular lymphatic glands was the only constant prodromal symptom; this swelling also could often be plainly seen, even as late as the third week after the beginning of the disease. Suppuration of the lymphatic glands has never been observed.

Further anatomical disturbances, especially a marked participation of the digestive mucous membrane, or an affection of the kidneys, as with scarlet fever, do not occur with normal rubeola. The urine was, in all cases in which I carefully examined it, perfectly normal, and at all events free from albumen, only the chlorides being present in excess. Emminghaus observed in one case of an adult with rubeola (together with other unusual symptoms pointing to a vaso-motor neurosis) a light albuminuria during the time of the exanthem.

As a rule the exanthem is the first, or at least among the first symptoms of disease; its appearance in a normal case is never preceded by a prodromal stage of any long duration, as, *e.g.*, in measles, where it lasts three days. If, therefore, the eruption shows itself during a feverish condition of some duration, the case may be put down as an anomalous one.

The friends of the patients often report that these had begun to show symptoms emanating from the mucous membranes, as early as from half a day to a day, rarely earlier (Emminghaus), before the first spots were noticed; more rarely we hear that fever with its associate manifestations set in just before the eruption. According to these reports, and to experiences in cases which could be observed as early as during the stage of incubation, the beginning of the eruption and that of the fever, in the cases at least where there was any, must have been separated by only a very short interval, so short that they might very properly be regarded as coincident. In normal cases of rubeola a prodromal stage of more than at most a half day cannot be assumed; the contrary constitutes an anomalous case, and proves that it is either a complication or a secondary rubeola, or evinces at least an abnormally great sensibility on the part of the children attacked. For the most part, however, no increase of temperature at all is noticed at the beginning of the exanthem, and it is improbable, according to the course of disease as stated, that any such was present before the beginning of the observation. Since, now, the assumption of a prodromal stage relates only to the existence of a feverish condition before the exanthem, the question in regard to it is in these cases, and according to my observations in the majority of cases of rubeola, a thoroughly idle one.

As regards the commencement of the eruption, I could not notice, in cases to which I was called early, any appreciable initial erythematous reddening of the skin, such as is at times found in the beginning of the acute exanthems. Feverish children, perspiring under warm feather-beds, were the only ones to manifest the moderate injection of the skin natural under such circumstances. Emminghaus describes a barely perceptible, slightly reddish coloration of the skin, like erythema, from which within a few hours the characteristic roseolæ were produced.

As regards the eruption of the exanthem upon the different parts of the body, rubeola follows the same law as measles and variola. In all regular cases the face and scalp are primarily attacked, and the eruption spreads gradually from here to the other parts of the body, first to the trunk and arms, finally to the lower half, especially the legs. This manner of extension presupposes the same relations of the specific contagion to the vaso-motor nerves which exist in the other acute exanthems. As an unimportant anomaly it may possibly happen that the manner of extension will deviate slightly from that stated; thus the face in particular may be only subsequently affected.

The development of the individual roseolæ requires but a very short time. I have frequently been able to convince myself beyond a doubt that no trace of the eruption had been present a half day before the appearance of the well-marked infiltrated red spots. The spread of the exanthem over the body is correspondingly rapid; it takes place, according to its intensity, in from one to two days. It therefore happens with tolerable frequency that the maxima of its development occur at varying times upon the different parts of the body. I have repeatedly noticed how the exanthem was at its height on the face, neck, and upper part of the trunk, while on the extremities, especially upon their lower portions, scarcely any suggestion of the spots was present. And when at last, after from twelve to twenty-four hours, they had reached here also the acme of their growth (usually fainter than on the face), there was then often but little still to be seen upon the parts first affected; the face was even at times already quite pale before the maximal development upon the extremities had been attained. It is very different in measles, with its essentially simultaneous maximum over the whole body. The different behavior of rubeola may, I think, be partially explained by the absence of fever, causing necessarily a diminished congestion of the skin. This congestion usually disappears entirely from all parts of the body within two or three days after its commencement upon the face. The duration of the maximum upon separate parts of the skin is much shorter than in measles; it requires perhaps at most a half day, frequently only a few hours.

The course of the temperature in rubeola is also very different from that in measles. In most cases, at least at the time during which I observed my patients, I failed to find any increase, unlike Emminghaus, who found it as a constant thing. If, therefore, any increase existed, it must have been present before my examination, and have already disappeared soon after the outbreak of the exanthem upon the face, which, especially as the friends noticed none, is not very probable. The majority of cases have no fever during the whole course of the disease, and the presence of temporary fever, at any particular time, is usually due to alterations in the temperature of the house, which may induce, for the time, a slight elevation in the temperature of the patient also. In the minority of cases fever, at times considerable, may exist; for the temperature may rise from 2° to 4° Fahr. above what is normal, though usually only about 1.5° Fahr. The elevation of temperature is either only an initial one, disappearing by the second day of the disease, or it may endure on the second, and even on the third day, at about the original height, in which case it may be ended by a speedy crisis, or may gradually sink to a normal condition. By the time the most essential symptom of the disease, the exanthem, has reached its end, the temperature in ordinary cases has become normal again; and this usually occurs, as stated, before, though sometimes after, the initial elevation. In the normal course of the temperature in rubeola there is most certainly no maximal elevation immediately before the crisis, coinciding with a universal maximum of the exanthem as in measles. Thus we see that the course of the temperature during rubeola is a very varying one, for there occur cases with normal temperature throughout, cases with fever during the whole eruption, with a rapid initial increase and a defervescence with crisis or lysis, and finally, cases with an initial fever and a defervescence concluded before the expiration of the eruption. The difference from measles is sufficiently characteristic if we consider what has been stated under that head.

The course of the disease is, therefore, in the majority of cases somewhat as follows: after the patients have coughed and sneezed somewhat, and manifested slight photophobia for from a few hours to a day, one notices—either at once, or after the attention has been excited by a gradually increasing temperature—the beginning of the exanthem upon the face. While now the exanthem gradually spreads over the whole body, the temperature, if increased, becomes more or less speedily normal again. In cases with only slight fever, the general health is frequently not disturbed, though when the fever is more severe and permanent, slight indisposition may exist. Children generally object to staying in bed, and would even prefer to be out of doors. In normal rubeola no other local symptoms of disease occur except those mentioned, namely, slight catarrh, and at times some difficulty in swallowing, if much angina is present, and so also, some transitory and slight disturbance of the appetite. Any nervous symptoms, chills, heat, thirst, etc., occur only with fever, in the manner usual under such circumstances.

Although it may be affirmed with certainty, from the course and appearances of the disease, that measles and rubeola are two specifically distinct infectious diseases, yet this is rendered even more evident by the fact that the two diseases afford no mutual protection; both may occur in the same person within a few weeks of each other. Nor does rubeola protect from scarlet fever, or *vice versâ*. This fact is testified to most conclusively by the most different observers, and it can therefore be regarded as beyond question. Further proof is afforded by the epidemic nature of the disease, especially of our epidemic of rubeola in 1872, as also by the fact that in the different families and domestic circles infection always produced the same disease. Opposing testimony will be sought almost in vain, even from the advocates of the identity of measles and rubeola.

After the disappearance of the exanthem, or even before it, the slight symptoms of inflammation of the mucous membranes subside, either simply or with the formation of a moderate amount of mucus. Convalescence, as the rule, runs an undisturbed course.

Complications.

In several reports statements may be found in regard to complications of rubeola. It is possible that in such cases at times a confounding with measles has taken place; this possibility, however, does not invariably exist. Such complications were chiefly severe attacks of bronchitis and affections of the lungs of different natures, occasioned evidently by the specific catarrh of the upper air-passages. As regards sequelæ, Thierfelder observed a febrile œdema of the face, Emminghaus a similar disturbance upon the legs, Mettenheimer a naso-pharyngeal catarrh, permanent swelling of the tonsils, inflammation of the gums, etc.

Prognosis.

The prognosis of simple primary rubeola is thoroughly favorable; it is the lightest of the acute exanthems, as we see from its almost feverless course. Complications can, however, according to their severity, occasion grave disturbances, or even death. A light rubeola, secondary to some other disturbance, and casually appearing during its course, rarely exercises a lasting or in any way considerable influence upon the course of the first disease, and the prognosis of the same is therefore not affected.

TREATMENT.

The treatment of rubeola is restricted to a suitable regimen, protection against exposure, bland diet, and keeping in bed if fever exists, cool sponging for any annoying itching, keeping watch upon the catarrh of the air-passages and of the pharynx, since this may call for active interference if at any time it should become severe. Complications are to be treated according to their nature, all the more because they rarely occur during the rubeola, but rather make their appearance long after this has run its course.

SCARLATINA.

BIBLIOGRAPHY.

On the subject of Scarlatina, one may consult the works on pathology, skin diseases, and diseases of children, which are mentioned in the literature of measles; and among the works which appeared in the beginning of this century, the monograph of *Most* (Versuch einer kritischen Bearbeitung der Geschichte des Scharlach, Leipzig, 1826), which contains a good compilation of the older literature, as also the monographs of *Kreysig* (1802), *Struve* (1803), *Reich* (1810), *Pfeufer* (1819), *Berndt* (1820 and 1827), *Böhm* (1823), *Seifert* (1827), *Weisenberg* (1828), *Steimmig* (1828), and *Fischer* (1832), are especially to be recommended. Many single articles have been written on scarlatina; the older ones may be found compiled in the second volume of *Canstatt's* Handbuch der medicinischen Klinik (Erlangen, 1847), p. 99. Of the more recent I refer, without attempting to be exhaustive, to the following, though of course I have had access to only a portion of the original writings. Anonymous publications, discussions, etc., may be found in *Canstatt's* Jahresbericht 1842. I. p. 524. 1844. IV. p. 235 and 233. 1852. IV. p. 204.—*Virchow-Hirsch*, Jahresbericht 1870. II. p. 264. p. 259. p. 416.—*Schmidt's* Jahrb. der ges. Med. 4 Supplbd. p. 116. 11 p. 89. 18 p. 254. 25 p. 372. 39 p. 365. 153 p. 242.—Journal für Kinderkrankheiten 1 p. 76. 77. 10 p. 239. 15 p. 467. 40 p. 426. 56 p. 319. 59 p. 103.—Jahrbuch für Kinderheilkunde 1859. II. 1. H. p. 44. 1861. IV. p. 129.—Würtemb. Correspondenzblatt 1854. XXIV. p. 201. 1857. XXVII. p. 183.—Prager Vierteljahrschrift 1847. 14 Bd. p. 3.—Schweiz. Ztschr. f. Med. 1856. p. 249.—Arch. d. Vereins f. wiss. Heilkunde. II. 1866. p. 491.—*Abelin*, J. f. Kkh. 41 p. 113.—*Addison*, Cst. Jber. 1865. IV. p. 102.—*Adler*, Schm. Jb. 4. Supplbd. p. 371.—*Albu*, Berl. kl. Woch. 1872. p. 629.—*Alison*, J. f. Kkh. 5 p. 8. Schm. Jb. 47 p. 170. Pr. Vjschr. 10 p. 89 An.—*d'Alves*, J. f. Kkh. 9 p. 215.—*v. Ammon*, Clar. u. Rad. Btr. Bd. III. Anal. üb. Kkheiten. 11. H. p. 42. 1836.—*Anderson*, Schm. Jb. 83 p. 207.—*Anderssen*, Varges' Ztschr. 15 p. 386.—*Andral* und *Gavarret*, J. f. Kkh. 8. p. 57. —*Anison*, Schm. Jb. 103 p. 313.—*Arnold*, Schm. Jb. 103 p. 25.—*Arrigoni*, Schm. Jb. 133 p. 319.—*Asdale*, J. f. Kkh. 59 p. 100.—*Asmus*, Cst. Jber. 1842. I. p. 526.—*Barensprung*, Ueb. die Folge u. d. Verlauf epidem. Kkheiten.—Halle, 1854.—*Baginsky*, Centrbl. 1870. Nr. 32. p. 497.—D. Ztschr. f. pr. Med.

1874. Nr. 15. p. 123.—*Billard*, Oest. Jb. f. Päd. 1870. B. p. 157.—*Ballot*, V.-H. Jber. 1871. II. p. 245.—*Balman*, Ctrbl. 1866. p. 256. Schm. Jb. 133 p. 318.—*Barach*, Schm. Jb. 4 Suppl. p. 365.—*Barclay*, Schm. Jb. 135 p. 239. —V.-H. Jber. 1871. II. p. 247.—*Barker*, Schm. Jb. 133 p. 317.—*Barnes*, Journ. f. Kkh. 5 p. 453.—Ibid. 16 p. 154.—*Bartels*, Virch. Arch. 21 p. 69.—*Barthez*, Schm. Jb. 13 p. 129.—*Bushan*, J. f. Kkh. 29 p. 198.—*Bauer*, Varg. Ztschr. 13 p. 97.—*Baumgärtner*, Cst. Jb. 1850. IV. p. 139.—*Bayer*, Arch. d. Heilk. IX. 1868. p. 136.—*Bayles*, Jbch. f. Khkde. 1874. VII. p. 226.—*Beale*, Ztschr. f. Parasitenk. III. p. 252. 1872.—*Becker*, Cst. Jb. 1846. IV. p. 131. —*Becker-Laurich*, V.-H. Jber. 1866. II. p. 246.—*Becquerel*, Séméiotique des urines. Paris, 1841. Schm. Jb. 36 p. 114.—*Bedgie*, Pr. Vtljschr. 40 p. 72.—*Bednar*, J. f. Kkh. 20 p. 384.—*Begbie*, Schm. Jb. 78 p. 180. Cst. Jber. 1849. IV. p. 201.—*Behrend*, J. f. Kkh. 12 p. 161. 44 p. 231 u. p. 455 Anm.—*Belitz*, Schm. Jb. 3 p. 24.—*Bell*, Schm. Jb. 75 p. 312.—Journ. f. Kkh. 56 p. 308 u. Berl. kl. Woch. 1871. Nr. 1. p. 10.—*Bennet*, Cst. Jb. 1851. IV. p. 143. 145.—1852. IV. p. 203.—1855. IV. p. 247.—*Berend*, Schm. Jb. 59 p. 296.—*Berg*, Würt. Corresp. XXIV. 1854. p. 85.—*Berkun*, Schm. Jb. 17 p. 43.—*Berndt*, Bemerk. üb. das Scharl. u. s. w. Greifswald, 1827.—*Berton*, J. f. Kkh. 1 p. 381.—*Besnier*, Schm. Jb. 140 p. 313.—*Betke*, Schm. Jb. 145 p. 190.—*Betz*, Journ. f. Kkh. 16 p. 386.—Schm. Jb. 112 p. 147.—Memorab. 1869. XIV. p. 193.—Jb. 1872. XVII. p. 141.—*Bidder*, Gers. u. Jul. Mag. 26 p. 56.—*Biefel*, Berl. kl. Woch. 1867. p. 13.—*Bierbaum*, J. f. Kkh. 45 p. 57.—*Biermer*, Würzb. Verh. Sitzb. f. 1859. p. 27. X. 1860 und Virch. Arch. 19 p. 537.—*Binz*, Jb. f. Khkunde 1871. IV. p. 103.—*Bird*, Schm. Jb. 49 p. 312.—*Blache*, Arch. f. Dermatol. 1870. II. p. 615.—*Blanche*, J. f. Kkh. 7 p. 312.—*Blanckaert*, V.-H. Jber. 1868. II. p. 257.—*Blaschko*, J. f. Kkh. 28 p. 155.—*Blondeau*, Oest. Jb. f. Päd. 1871. B. p. 131.—*Böning*, Deutsche Klinik 1870. Nr. 30–33.—*Bohn*, Jbch. f. Khkunde 1869. II. p. 448. 1870. III. p. 46.—*Bokai*, Jbch. f. Khkde. V. 1862. p. 85.—*Bonnassies*, J. f. Kkh. 4 p. 230.—*Bornhaupt*, Rig. Btrg. z. Hkde. 1855. III. p. 125.—*Boss*, Cst. Jb. 1857. IV. p. 222. *Botrel*, Schm. Jb. 59 p. 281.—*Bouchut*, J. f. Kkh. 39 p. 112. Cst. Jb. 1862. IV. p. 125.—*Bouragne*, Cst. Jb. 1859. IV. p. 224. *Bramwell*, V.-H. Jber. 1870. II. p. 264. Oest. Jb. f. Päd. 1871. B. p. 80.—*Brandt*, Schm. Jb. 22 p. 360.—*Brattler*, Btrge. zur Urologie. München, 1858. Schm. Jb. 104 p. 12.—*Braun*, Schm. Jb. 29 p. 307. u. 309. Jb. 30 p. 149.—*Bretonneau*, Schm. Jb. 6 p. 53.—*Brokmann*, Schm. Jb. 18 p. 185.—*Brosius*, Schm. Jb. 133 p. 336.—*Brotherston*, J. f. Kkh. 22 p. 124.—*Brown*, J. f. Kkh. 6 p. 355. Pr. Vjschr. 30 p. 86 d. An. Cst. Jb. 1844. IV. p. 234.—*Brun*, Pr. Vjschr. 5 p. 86 d. An.—*Brunton*, V.-H. Jb. 1871. II. p. 245.—*Buchanan*, Schm. Jb. 142 p. 153.—*Buchner*, *D. Hahnemann's* Heilg. u Verhütg. etc. 1844.—*Budd*, V.-H. Jber. 1869. II. p. 244.—*Bürger*, Wurt. Corr. 1871. 29.—*Bürkner*, Schm. Jb. 61 p. 291.—*Büttner*, Schm. Jb. 37 p. 11.—*Bulley*, Schm. Jb. 65 p. 171.—*Burke*, Schm. Jb. 18 p. 186.—*Burrowes* u. *Kirkes*, J. f. Kkh. 22 p. 112.—*Buttura*, Schm. Jb. 95 p. 121.—*Cabot*, J. f. Kkh. 18 p. 449.—*Camerer*, Würt. Corr. 1842.

p. 31. 1844 p. 90.—*Carpenter*, V.-H. Jb. 1871. II. p. 247.—*Carrière*, Cst. Jb. 1843. III. p. 218.—*Ceysens*, Cst. Jb. 1860. IV. p. 135.—1861. IV. p. 201. —*Charlton*, J. f. Kkh. 9 p. 190 —*Chavasse*, J. f. Kkh. 31 p. 301.—*Chomel*, Pr. Vjschr. 15 p. 64 d. An. J. f. Kkh. 6 p. 126.—*Chrastina*, Schm. Jb. 108 p. 208. —*Clark*, Schm. Jb. 34 p. 48.—Ibid. 131 p. 31.—*Clarus*, Schm. Jb. 3 p. 214.—*Clemens*, Schm. Jb. 86 p. 199. J. f. Kkh. 34 p. 1.—*Cless*, Würt. Corr 1847. p. 209.—1854. p. 357.—*Cockle*, Schm. Jb. 88 p. 211.—*Cohen*, Schm. Jb. 7 p. 275.—Jb. 2 Supplbd. p. 112.—Cst. Jb. 1841. p. 55.—Jb. 1842. I. p. 523.—*Cohn*, Hydrotherapie d. Scharl. Berl., 1862.—*Cole*, Cst. Jb. 1862. IV. p. 128.—*Coley*, Cst. Jb. 1848. IV. p. 139.—*Constant*, Schm. Jb. 4 p. 333.—Jb. 6 p. 108. —Jb. 14 p. 92.—*Copeman*, Cst. Jb. 1842. II. p. 40.—V.-H. Jb. 1871. II. p. 247.—*Cordwent*, V.-H. Jb. 1870. II. p. 260.—*Cormack*, Pr. Vjschr. 27 p. 101 d. An.—Cst. Jb. 1850. IV. p. 139.—*Corrigan*, Pr. Vjschr. 3 p. 97 d. An.—Jb. 6. p. 177 d. An.—J. f. Kkh. 5 p. 255.—*Corson*, V.-H. Jber. 1871. II. p. 249.—*Coulson*, J. f. Kkh. 34 p. 446.—Jb. 47 p. 424.—*Coze* u. *Feltz*, Schm. Jb 154 p. 239. —*Cramer*, Rust's Magazin, 25 p. 556.—*Cremen*, Cst. Jb. 1863. IV. p. 130.—*Cummins*, Cst. Jb. 1865. IV. p.102.—*Dühne*, cit. in Cst. Jb. 1851. IV. p. 145. —*Danielssen*, V.-H. Jber. 1868. II. p. 255.—*Davaine*, Cst. Jber. 1855. IV p. 247.—*Dechaux*, Cntrbl. 1868. p. 224.—*Deininger*, D. Arch. f. kl. Med. VII. p. 587.—*Deiters*, D. Klin. 1859. 12. 31. 32. 34.—*Delvaux*, Cst. Jb. 1852. IV. p. 207.—*Denizet*, V.-H. Jb. 1868. II. p. 254.—*Deperet-Muret*, Cst. Jb. 1851. IV. p. 144. J. f. Kkh. 15 p. 475.—*Deutschert*, Schm. Jb. 4 Suppbld. p. 876.—*Deval*, Schm. Jb. 67 p. 229.—*Devaucleroy*, V.-H. Jb. 1871. II. p. 250. *Dewar*, Schm. Jb. 11 p. 165.—*Dickinson*, Schm. Jb. 115 p. 305.—*Dikinson*, Jbch. f. Khknde. VII. 2. H. p. 96. 1864.—*Dittrich*, Pr. Vjschr. 12. Orig. p. 186.—*Dobigny*, J. f. Kkh. 7 p. 311.—*Dopfer*, Beob. u. Abh. öst. Aerz. 1823. III. p. 433.—*Drake*, Schm. Jb. 90 p. 373.—*Druitt*, Bay. Intell. 1871. 14.—*Duchek*, Pr. Vjschr. 37. Or. p. 95.—*Duchesne-Duparc*, Schm. Jb. 63 p. 43.—*Duckes*, Schm. Jb. 99 p. 55.—*Dührssen*, Schm. Jb. 8 p. 290.—*Duncome*, Cst. Jb. 1846. IV. p. 131.—*Duriau*, Schm. Jb. 94 p. 283.—*Dyes*, Küchenm. Ztschr. 1867. VI. N. F. p. 390. J. f. Kkh. 54 p. 268.—*Easton*, V.-H. Jber. 1870. II. p. 259.—*Ebert*, Charité Ann. 1850. p. 147. p. 770.—Schm. Jb. 140 p. 139.—*Edwards*, Cst. Jbr. 1862. IV. p. 129.—*Eichmann*, Varges Ztschr. VIII. p. 52. 1855.—*Eiselt*, Schm. Jb. 19 p. 32.—*Eisenmann*, Würzb. Verh. 1852. III. p. 209.—Schm. Jb. 21 p. 139.—Cst. Jb. 1850. IV. p. 137. —Ibid. 1857. IV. p. 221 Anm.—*Eisenschitz*, Wien. med. Presse. VII. 1864. p. 1084.—Jbch. f. Khknde. 1871. IV. p. 242.—*Ellinger*, Würt. Corr. 1854. XXIV. p. 176.—*Elsässar*, Schm. Jb. 10 p. 322.—*Elwert*, Rust's Magaz. 31 p. 110.—*Epting*, Würt. Corr. 1855. XXV. p. 79.—*Erhard*, Ueb. d. äuss. Anw. kalt. Wass. im. Sch. Nördlingen, 1824.—*Eulenberg*, Schm. Jb. 108 p. 211.—*Eulenberg*, Berl. kl. Woch. 1869. VI. p. 435.—*Faber*, Würt. Corr. 1852. XXII. p. 221. 1856. XXVI. p. 334.—*Fagge*, Cntrbl. 1868. p. 272.—*Fallot*, Pr. Vjschr. 74 p. 41 d. An.—J. f. Kkh. 33 p. 121.—*Feitel*, Schm. Jb. 108 p. 209. —*Fenini*, V.-H. Jber. 1872. II. p. 253.—*Fenwick*, Schm. Jb. 133 p. 317.

—*Fergus*, V.-H. Jbr. 1869. II. p. 244.—*Finckenstein*, Küchenm. Ztschr. 1862. N. F. I. p. 94. 139.—*Fischer*, Cst. Jber. 1846. IV. p. 131.—Schm. Jb. 81 p. 56.—*Fitzpatrick*, Schm. Jb. 4 Suppl. p. 372.—Cst. Jber. 1852. IV. p. 204.—*Fleischmann* (Erlangen). Schm. Jb. 10 p. 19.—*Fleischmann*, Jbch. f. Khkde. 1870. III. p. 466.—Ibid. 1871. IV. p. 166. p. 174.—Arch. f. Dermat. 1872. IV. p. 223.—*Förster*, Diss. Leipzig, 1859.—Pr. Vjschr. 84 p. 7 u. 65 d. Orig. —Jbch. f. Khkde. 1868. I. p. 121. p. 133. 1872. V. p. 325.—*Fox*, Cst. Jber. 1864. IV. p. 124.—*Francis*, Schm. Jb. 99, 57.—Cst. Jber. 1861. IV. p. 200.—*Fraser*, J. f. Kkh. 59. p. 119.—*Frerichs*, Die Brightsche Nierenkr. Braunschw. 1851.—*Friese*, Pr. Vjschr. 6 p. 80 d. An.—*Frölich*, Schm. Jb. 40 p. 151.—Beob. u. Abh. öst. Aerz. 1828. VI. p. 352.—*Fuller*, Schm. Jb. 142 p. 154.—*Gairdner*, J. f. Kkh. 20 p. 126.—*Gajasy*, Berl. kl. Woch. 1870. p. 616. *Garnier*, Cst. Jber. 1859. III. p. 210.—*Gaupp*, Würt. Corr. 1854. XXIV. p. 13.—1856. XXVI. p. 105.—*Gauster*, Schm. Jb. 99 p. 59.—*Geertsema*, J. f. Kkh. 3 p. 05.—*Geissler*, Küch. Ztschr. 1862. I. p. 404.—D. Vjschr. f. öff. Geshpfl. III. p. 47.—*Gerhardt*, D. Arch. f. kl. Med. XII. p. 1. *Giersing*, V.-H. Jber. 1871. II. p. 250.—*Gillespie*, J. f. Kkh. 23 p. 152.—Cst. Jber. 1853. IV. p. 167.—Ibid. 1862. IV. p. 124.—*Girard*, Cntrbl. 1865. p. 863. *Gläser*, Schm. Jb. 80 p. 234.—*Godelle*, J. f. Kkh. 3 p. 159.—Pr. Vjschr. 3 p. 97 d. An.—*Goos*, Schm. Jb. 150 p. 319.—*Gorée*, Cst. Jber. 1842. I. p. 524.—*Gouzée*, Schm. Jb. 3 p. 339.—*Graf*, Schm. Jb. 18 p. 187.—*Grantham*, Schm. Jb. 107 p. 293.—*Graves*, Klin. Beob. D. v. Bressler. Leipz., 1843. Pr. Vjschr. 3 p. 95 d. An.—Cst. Jber. 1843. III. p. 217.—Ibid. 1844. IV. p. 234.—Oest. med. Woch. 1847. 3. Qu. p. 906.—*Gregory*, J. f. Kkh. 4 p. 46.—Vorles. üb. d. Ausschlagsfieber. D. v. Helfft. Leipzig, 1845. p. 126.—*Guéneau de Mussy*. V.-H. Jber. 1871. II. p. 249.—*Günsburg*, Schm. Jb. 68 p. 51.—Ibid. 83 p. 206.—*Guérilin*, Schm. Jb. 4 Supplbd. p. 372.—*Guersant* (Vater), J. f. Kkh. 3 p. 42.—Encycl. d. Med. Wiss. 1833. X. p. 440.—*v. Gunz*, Jbch. f. Khkunde 1862. V. p. 161.—*Gutherz*, Cst. Jber. 1864. IV. p. 128.—*Gutmann*, Ueb. d. Gesetze d. Epid. d. Scharl. Diss. Würzb., 1859.—*Guy*, Schm. Jb. 41 p. 38.—*Härlin*, Würt. Corr. 1854. XXIV. p. 116—1861. XXXI. p. 155.—*Hale*, Schm. Jb. 73 p. 201.—*Hallier*, Jbch f. Khkde. 1869. II. p. 169. —*Hamburger*, Pr. Vjschr. 69. Orig. p. 24.—*Hambursin*, J. f. Kkh. 35 p. 214. u. 36. p. 11.—*Hamel*, Cst. Jber. 1842. II. p. 40.—*Hamilton*, Schm. Jb. 9 p. 180.—18 p. 184.—*Hamilton*, Cst. Jber. 1863. IV. p. 131.—*Hammond*, Schm. Jb. 7 p. 26.—*Hardy*, V.-H. Jber. 1868. II. p. 636.—*Hare*, J. f. Kkh. 58 p. 152.—*Harley*, V.-H. Jber. 1871. II. p. 248.—J. f. Kkh. 58 p. 153.—*Harrinson*, Cst. Jber. 1864. IV. p. 125.—*Hauff*, Würt. Corr. 1855. XXV. p. 121. —1856. XXVI. p. 122.—1862. XXXII. p. 324.—1863. XXXIII. p. 333. 339. 347. 351.—*Hauner*, Schm. Jb. 69 p. 200.—Ibid. 73 p. 198.—J. f. Kkh. 17 p. 2. —Ibid. 49 p. 281.—*Hawkins*, J. f. Kdkh. 17 p. 65.—*Haydon*, Cst. Jber. 1854. IV. p. 150.—*Hebra*, Schm. Jb. 107 p. 120. 112 p. 252.—*Hecht*, V.-H. Jber. 1868. II. p. 254.—*Heckford*, Arch. f. Derm. 1869. I. p. 274.—*Heim*, Rust's Magaz. 28 p. 72.—*Heim*, Würt. Corr. 1864. XXXIV. p. 195.—*Heine*, Schm.

Jb. 8 p. 210.—*Helfer*, Oest. Jahrbuch für Päd. 1870. B. p. 223.—*Helfft*, Journal für Kinderkrankheiten 1 p. 10.—Ibid. 12 p. 359.—Cst. Jber. 1849. IV. p. 199.—*Henderson*, Schm. Jb. 102 p. 93.—*Hennig*, Schm. Jb. 76 p. 369. Jbch. f. Khkde. 1871. IV. p. 78.—*Henoch*, Berl. kl. Woch. 1865. p. 121.—Ibid. 1868. p. 93.—Ibid. 1873. p. 593.—Beitr. z. Khknde. N. F. 1868.—*Hertel*, Schm. Jb. 15 p. 291.—*Hervieux*, Schm. Jb. 137 p. 309.—*Heslop*, V.-H. Jber. 1870. II. p. 263.—*Hewitt*, J. f. Kkh. 30 p. 321.—*Heyder*, Diss. Berl., 1870. V.-H. Jber. 1871. II. p. 246.—*Heyfelder*, Schm. Jber. 8 p. 103. 11 p. 217. 24 p. 238. 32 p. 304. Berl. kl. Woch. 1868. p. 444.—Stud. im Geb. d. Heilw. II. p. 68.—*Hicks*, Schm. Jb. 153 p. 168.—*Higginbotham*, Petersb. Med. Ztschr. N. F. I. 1870. p. 344.—*v. Hildenbrand*, Ueb. d. anst. Typhus. 2 Aufl. Wien, 1814. p. 150.—*Hillier*, J. f. Kkh. 39 p. 385. —V.-H. Jber. 1868. II. p. 645.—Cst. Jb. 1862. IV. p. 126.—*Hingeston*, Schm. Jb. 7 p. 27.—*Hirsch*, Schm. Jb. 4. Spplbd. p. 376.—*Hirschsprung*, V.-H. Jber. 1871. II. p. 611.—*Hochstetter*, Schm. Jb. 94 p. 181.—*Hölder*, Würt. Corr. 1856. p. 25.—*Höring*, Würt. Corr. 1844. p. 263. 1854. p. 160. 1854. p. 332. 1856. p. 152. 1856. p. 304.—*Hoffmann*, Pr. Vjschr. 5 p. 86 d. An.—*Hofmann*, Ztschr. f. Parasitenk. III. 1872. p. 105. —*van Holsbeck*, Schm. Jb. 99 p. 59.—*Holscher*, Pr. Vjschr. 3 p. 96 d. An.—*Holst*, V.-H. Jber. 1871. II. 250.—*Holst u. Faye*, Norsk Mag. for Laegevid. 1873. p. 126.—*Hood*, J. f. Kkh. 5 p. 264. 31 p. 304.—Schm. Jb. 99 p. 52.—Arch. f. Derm. I. p. 460. 1869.—*Hoppe*, Arch. d. V. f. gem. Arb. II. 1856. p. 153.—*Horn*, Schm. Jb. 18 p. 187. 124 p. 270.—*Hornemann*, V.-H. Jber. 1871. I. p. 457.—*Horning*, Cst. Jber. 1861. IV. p. 201.—*Howard*, Schm. Jb. 34 p. 49.—*Huber*, D. Arch. f. kl. Med. 1871. VIII. p. 422.—*Hübler*, Diss. Leipzig, 1861.—*Hüter*, Diss. Marburg, 1858.—*Huguenin*, Pathol. Unters. Zürich, 1869. p. 63.—*Hulke*, V.-H. Jber. 1872. II. p. 253.—*Huppert*, Schm. Jb. 119 p. 23.—*Hutchinson* u. *Jackson*, Cst. Jber. 1863. IV. p. 132.—*Hynes*, V.-H. Jber. 1870. II. p. 264.—*Ilisch*, Med. Zeitg. Russl. 1851. Schm. Jb. 71 p. 317.—*Isambert*, Schm. Jb. 95 p. 185.—*Jacobs*, Berl. kl. Woch. 1865. p. 285.—*Jadioux*, J. f. Kkh. 5 p. 278.—*Jaffe*, Schm. Jb. 113 p. 102.—*Jahn*, Rust's Magaz. 28 p. 69.—*Jenner*, J. f. Kkh. 55 p. 1.—Arch. f. Derm. 1870 II. p. 593. —Jb. f. Khkunde. III. p. 348.—V.-H. Jber. 1870. II. p. 261.—*Joël*, Hufel. Journ. 1842. April. p. 3.—*Johnson*, J. f. Kkh. 22 p. 130. Ctrbl. 1871. p. 26.—*Jones*, Varges Ztschr. 1852. 5 Bd. p. 384.—J. f. Kkh. 22 p. 117.—*Just*, Schm. Jb. 109 p. 333.—*Kapff*, Würt. Corr. 1852. XXII. p. 234.—*v. Karajan*, Med. Jahrb. XXI. Jahrg. 2. Bd. p. 9 u. 22. 1865.—*Kaurin*, V.-H. Jber. 1868. II. p. 252.—*Keber*, Schm. Jb. 42 p. 184.—*Kelso*, J. f. Kkh. 8 p. 261.—*Kennedy*, J. f. Kkh. 2 p. 119. Ibid. 6 p. 464. Ibid. 25 p. 417.—Pr. Vjschr. 50 p. 78 d. An.—Cst. Jber. 1854. IV. p. 150.—Ibid. 1862. IV. p. 126.—Ibid. 1863. IV. p. 133.—V.-H. Jber. 1871. II. p. 246.—*Kern*, Würt. Corr. 1854. XXIV. p. 7.—*Kerr*, V.-H. Jahresber. 1870. II. p. 263.—*Kettle*, V.-H. Jber. 1866. II. p. 246.—*Kieser*, Schm. Jb. 6 p. 214.—*King*, Schm. Jb. 73 p. 202.—*Kinglake*, Pr. Vjschr. 40 p. 75 d. An.—*Kirkpatrick*, J. f. Kkh. 7 p.

Ctrbl. 1871. p. 655.—*Raffi*, V.-H. Jber. 1872. II. p. 253.—*Ranke*, Jbch. f. Khkde. 1869. II. p. 43.—*Ratter*, Schm. Jb. 1 Suppl. p. 178.—*Ravn* u. *Aarestrup*, V.-H. Jb. 1867. II. p. 6.—*Redenbacher*, Cst. Jb. 1862. III. p. 252. —Jbch. f. Khkde. 1861. IV. Beil. p. 16. Schm. Jb. 125 p. 18.—*Reeves*, Cst. Jb. 1845. IV. p. 170.—*Reevis*, J. f. Kkh. 35 p. 320.—*Rehn*, Jbch. f. Khkde. 1869. p. 439.—*Reisinger*, Oest. med. Jbch. 59. Bd. 1847. 4 Qu. p. 129.—*Reisland*, Diss. Leipzig, 1870.—*Renz*, Würt. Corr. 1854. XXIV. p. 225.—*Retzius*, Schm. Jb. 116 p. 318.—*Richardson*, J. f. Kkh. 22 p. 253.—Ibid. 48 p. 295.—Schm. Jb. 116 p. 125.—*Richter*, Würt. Corr. 1856. XXVI. p. 248.—*Richter*, Schm. Jb. 159 p. 207.—*Rieken*, Schm. Jb. 42 p. 113.—*Riess*, Reich. u. Dubois's Arch. 1872. p. 240.—*Ringer*, J. f. Kkh. 46 p. 99.—Cst. Jber. 1862. IV. p. 126. 127.—Schm. Jb. 143 p. 154.—*Ritter*, Cst. Jber. 1864. VII. p. 109.—*Robert*, Cst. Jber. 1850. IV. p. 140.—*Roberts*, Arch. f. Derm. 1871. III. p. 577.—*Robinson*, V.-H. Jber. 1870. II. p. 264.—*Roe*, J. f. Kkh. 21 p. 234.—*Röbbelen*, Cst. Jber. 1861. IV. p. 204.—*Rösch*, Schm. Jb. 5 p. 12.—Jb. 7 p. 71.—Würt. Corr. 1837. VII. p. 389.—Ibid. 1839. IX. p. 329.—Cst. Jb. 1844. IV. p. 230.—*Röser*, Schm. Jb. 47 p. 295. Pr. Vjschr. 8 p. 51 d. An.—Würt. Corr. 1854. XXIV. p. 209.—Ibid. 1855. XXV. p. 33.—*Roger*, J. f. Kkh. 4 p. 65. 255.—Ibid. 51 p. 86.—Cst. Jber. 1863. IV. p. 132.—*Rohde*, Med. Jahrb. 1865. XXI. 2. Bd. p. 13 d. An.—*Romberg*, Schm. Jb. 10 p. 60.—J. f. Kkh. 1 p. 278.—*Rosenstein*, Virch. Arch. 14 p. 132.—*Rosenthal*, Schm. Jb. 8 p. 46.—*Rostan*, Pr. Vjschr. 3 p. 96 d. An.—*Rothe*, Berl. kl. Woch. VII. p. 292. 1870.—*Rothe*, Pr. Vjschr. 1845. 8. Bd. p. 53 d. An.—*Routh*, J. f. Kkh. 13 p. 136.—*Rovida*, Pr. Vjschr. 116 p. 45 d. An.—*Rowland*, Cst. Jber. 1848. IV. p. 139.—*Rühle*, Kehlkopfkh. Berlin, 1861.—*Rupprecht*, Wien. med. Woch. 1862. p. 435.—*Russegger*, Oest. m. Jb. 1848. 63. Bd. 4 Qu. p. 277.—*Ryan*, Cst. Jb. 1852. IV. p. 204.—*Salzmann*, Würt. Corr. 1860. p. 77.—*Samter*, Königsb. med. Jb. 1864. IV. p. 147.—*Sander*, Cst. Jb. 1850. IV. p. 137.—*Sanderson*, Jf. Kkh. 36 p. 460.—*Sandwith*, Schm. Jber. 1 p. 14.—*Sansom*, Schm. Jb. 148 p. 12. 153 p. 167.—V.-H. Jb. 1871. II. p. 246.—*Santlus*, Jf. Kkh. 22 p. 300. 302.—*Schallenmüller*, Würt. Corr. 1843. XIII. p. 340. 1856. XXVI. p. 144.—*Schauenburg*, Arch. d. V. f. wiss. Hk. 1860. IV. p. 195.—*Schiefferdecker*, Ueb. d. Einfl. d. ac. Htausschl. auf d. Kdrstrbl. Königsberg, 1870.—*Schlesier*, Schm. Jb. 31 p. 52.—*Schmidt*, Berl. kl. Woch. 1870. VIII. 48.—*Schneck*, Schm. Jb. 96 p. 309.—Cst. Jber. 1859. IV. p. 126.—*Schneemann*, Heilg. des Sch. Hannover, 1848.—*Schnizlein*, Das Schfieber, etc. München, 1851.—*Schöpf*, J. f. Kkh. 9 p. 5.—*Schuberg*, Schm. Jb. 114 p. 400.—*Schüz*, Würt. Corr. 1854. XXIV. p. 209.—*Schwandner*, Würt. Corr. 1856. XXVI. p. 168.—*Schwartz*, Rig. Beitr. z. Heilk. 1851. I. p. 530.—*Schwarz*, Schm. Jb. 19 p. 31.—*Schwarz*, Diss. Greifswald, 1860.——*Schwarz*, Wien. med. Presse 1871. Nr. 42.—Arch. f. Derm. 1872. IV. p. 443.—*Schwarz*, V.-Jber. 1868. II. p. 256.—*Schweich*, Pr. Vjschr. 5 p. 85 d. An.—*Scoutetten*, Schm. Jb. 102 p. 313.—Cntrbl. 1868. p. 799.—*Sedgwick*, Cst. Jber. 1851. IV. p. 144.—*Sée*, Schm. Jb. 102 p. 109.—*Seifert*, Nosol. Bem.

über Scharl. Griefsw., 1827.—Rust's Magaz. 33 p. 395.—*Seitz*, Cst. Jber. 1851. IV. p. 143.—Bay. Intell. 1873. p. 757.—*Senfft*, Würzb. med. Zeitg. 1864. V. p. 170.—*Seux*, Memorab. 1865. X. p. 21.—*Shepherd*, Med.-chir. Rundschau. 1868. IX. Jahrg. 1. Bd. p. 109. Ctrbl. 1868. p. 272.—*Shingleton Smith*, Arch. f. Derm. 1871. III. p. 271.—*Sicherer*, Schm. Jb. 35 p. 335.—*Siedner*, Pr. Vjschr. 2 p. 122 d. An.—*Siemon-Dawosky*, Schm. Jb. 108 p. 207.—*Simon*, Pr. Vjschr. 1 p. 113 d. An.—Schm. Jb. 34 p. 147.—Ibid. 39 p. 9.—*Simon*, John. Oest. Jb. f. Päd. 1871. B. p. 5.—*Simon* (Hamburg). Arch. f. Derm. 1873. V. p. 103.—*Skoda*, Schm. Jb. 118 p. 34.—*Smith*, V.-Jber. 1871. II. p. 250.—J. f. Kkh. 59 p. 119.—*Smoler*, Pr. Vjschr. 82 p. 93 d. Or.—*Snow*, Schm. Jb. 47 p. 172.—Cst. Jb. 1844. IV. p. 235.—Pr. Vjschr. 10 p. 90.—*Solbrig*, Schm. Jb. 29 p. 310.—*Sommola*, Jbch. f. Khkde. 1868. I. p. 227.—*Spadafora*, Schm. Jb. 17 p. 294.—*Spender*, V.-H. Jb. 1870. II. p. 264.—Arch. f. Derm. III. p. 270.—*Spielmann*, Diss. Strassb., 1856. Schm. Jb. 93 p. 9.—*Spiess*, s. die Jahresber. der Stadt Frankfurt a. M.—*Spiro*, Schm. Jb. 70 p. 97. *Stäger*, J. f. Kkh. 20 p. 1.—*Stannius*, Schm. Jb. 6 p. 142.—*Stark*, Schm. Jb. 18 p. 182.—*Steffen*, Jbch. f. Khkde. VIII. p. 166.—Ibid. N. F. IV. 1871. p. 317.—*Steinbacher*, Scharl. u. Mas. Augsb., 1865.—*Steinbeck*, Pr. Vjschr. 1 p. 113 d. An. u. 2 p. 122 d. An.—*Steiner*, Jbch. f. Khkde. 1868. I. p. 432.—*Steiner u. Neureutter*, Pr. Vjschr. 84 p. 92.—Ibid. 105 p. 79.—106 p. 60 u. 70.—*Steinthal*, Schm. Jb. 1. Spplbd. p. 288. Ibid. 55 p. 300.—J. f. Kkh. 44 p. 326. 51 p. 44.—*Stevens*, Schm. Jb. 38 p. 161.—*Stevenson*, J. f. Kkh. 59 p. 116.—*Stiebel*, J. f. Kkh. 33 p. 145 u. 285.—*Stiévenart*, Schm. Jb. 43 p. 113.—*Stratton*, J. f. Kkh. 6 p. 336.—*Studinsky*, Schm. Jb. 31 p. 51.—*Sturm*, Cst. Jber. 1850. IV. p. 140.—*Sutton*, Schm. Jb. 34 p. 50. 99 p. 56.—*Swenzizky*, Diss. Petersb., 1861.—*Swete*, J. f. Kkh. 14 p. 150.—*Swiney*, Arch. f. Derm. 1870. II. p. 614. —V.-H. Jber. 1870. II. p. 261.—*v. Sydow*, J. f. Kkh. 51 p. 144.—*Syme*, Cst. Jber. 1841. p. 57.—*Tait*, Schm. Jb. 151 p. 340.—*Taupin*, J. f. Kkh. 1 p. 378. *Taylor*, Schm. Jb. 43 p. 39.—Jb. 151 p. 340.—*Theurer*, Würt. Corr. 1844. p. 207.—*Thirial*, J. f. Kkh. 7 p. 311.—*Thomas*, Memorab. 1869. XIV. p. 215.—Arch. d. Heilk. 1869. X. p. 458.—Ibid. 1870. XI. p. 130 u. 449.—Jahrb. f. Khkde. 1869. II. p. 373.—Ibid. 1870. III. p. 85.—Ibid. 1871. IV. p. 1 u. 60.—*Thompson*, J. f. Kkh. 5 p. 310.—V.-H. Jber. 1869. II. p. 244.—1870. II. p. 4 u. 264.—*Thomson*, J. f. Kkh. 2 p. 74.—Schm. Jb. 96 p. 94.—*Thore*, Schm. Jb. 91 p. 89.—*Thoresen*, Pr. Vjschr. 116 p. 12 d. Anz.--V.-H. Jber. 1867. II. p. 278.—1872. II. p. 255.—*Todd*, J. f. Kkh. 12 p. 432.—Schm. Jb. 128 p. 252.—*Tolmatschew*, Jbch. f. Khkde. 1869. II. p. 220.—*Tott*, J. f. Kkh. 26 p. 419.—Varg. Ztschr. 1857. XI. p. 190.—*Tourtual*, Hufel. Journ. 1826, Dec. p. 3.—*Townsend*, V.-H. Jb. 1869. II. p. 244.—*Trapenard*, Cst. Jb. 1862. IV. p. 126.—*Tripe*, Pr. Vjschr. 44 p. 86.—Ibid. 47 p. 85. Schm. Jb. 87 p. 220.—*Trojanowsky*, Dorp. med. Ztschr. I. p. 297. III. p. 199. IV. p. 19.—*Trousseau*, Schm. Jb. 99 p. 53.—Cst. Jber. 1853. IV. p. 168.—1854. IV. p. 149.—1861. IV. p. 203.—J. f. Kkh. 3 p. 239. 13 p. 117. 15 p. 428. 21 p. 114. 29 p. 448. 30 p. 255.—Pr. Vjschr. 42 p. 74. 45 p. 78. 94 p. 60.—Med. Klin. des

Hôtel-Dieu.—*D. v. Culmann.* Würzb., 1866.—*Tüngel*, Klin. Mittheil. aus 1858, p. 27 u. Virch. Arch. 16 p. 360.—Kl. Mitth. aus 1859 p. 23. u. 165; aus 1860, p. 17.—*Turner*, Cst. Jber. 1861. IV. p. 201.—*Ullersperger*, Bay. Intell. 1871. p. 147.—*Veasey*, Arch. f. Derm. I. p. 462.—*Veit*, Würt. Corr. 1854. XXIV. p. 204.—*Veit*, Berl. kl. Woch. 1868. p. 452. 461.—*Virchow*, Rundschau. 1869. X. 4. Bd. p. 109 aus Ber. d. Natf. vers. zu Innsbruck.—*Vogel*, Würt. Corr. 1854. XXIV. p. 209.—*Vogel*, Petersb. med. Ztschr. 1868. XV. p. 118.—*Vogt*, V.-H. Jber. 1871. II. p. 250.—Med. Cntrlztg. 1873. p. 1240.—*Voigt*, Schm. Jb. 18 p. 187 u. 31 p. 57.—*Voit*, Jbch. f. Khkde. V. 1872. p. 266.—*Volz*, Schm. Jb. 67 p. 214.—*Vose*, Schm. Jb. Suppbld. 4 p. 370.—*Wagner*, Arch. d. Heilk. 1867. p. 262.—*Waidele*, Schm. 99 p. 58.—*Wallach*, Cst. Jb. 1844. IV. p. 233.—*Walz*, Schm. Jb. 78 p. 166.—Cst. Jb. 1852. IV. p. 205 und 1853. IV. p. 169.—*Wasastjerna*, V.-H. Jber. 1870. II. p. 265.—*Wasserfuhr*, V.-H. Jber. 1866. II. p. 562.—*Wassmann*, Cst. Jber. 1841. p. 57.—*Watts*, Sch. Jb. 4. Supplbd. p. 374.—*Weber* (London), Schm. Jb. 133 p. 332.—V.-H. Jber. 1866. II. p. 246.—Cntrbl. 1866. p. 720.—*Weber*, Varges Ztschr. 1858. XII. p. 89. 169.*273.—*Webster*, J. f. Kkh. 16 p. 151.—28 p. 403.—*Weineck*, Die Epid. d. St. Halle 1852.—1871. Halle, 1872.—*Weisse*, J. f. Kkh. 9 p. 369. 25 p. 96. 27 p. 52 u. 63.—*Weitenweber*, Schm. Jb. 12 p. 295.—*Welsch*, Bay. Intell. 1874. p. 26.—*Wendelboe*, Gers. u. Jul. Mag. 8 p. 188.—*West*, Pr. Vjschr. 39 p. 64.—*Wetzler*, Med.-chir. Zeitg. 1814. I. p. 126.—*Widerhofer*, Jbch. f. Khkde. 1860. III. p. 204 u. 276.—*Wildberg*, Schm. Jb. 6 p. 145.—*Wilks*, J. f. Kkh. 42 p. 122.—*v. Willebrand*, V.-H. Jber. 1870. II. p. 264.—*Williams*, Schm. Jb. 21 p. 139.—Cst. Jber. 1861. IV. p. 202.—*Willis*, Cst. Jber. 1842. I. p. 524.—*Willshire*, J. f. Kkh. 4 p. 457. 27 p. 408. *Winge*, V.-H. Jber. 1871. II. p. 250.—*Wintrich*, Memorab. 1867. XII. p. 203. *Wisshaupt*, Pr. Vjschr. 19 p. 47 u. 22 p. 101.—*Witt*, Schm. Jb. 133 p. 319.—*Wolff*, Arch. d. V. f. wiss. Hkde. 1866. II. p. 134.—*Wood*, Schm. Jb. 1. Suppl. p. 114.—Jf. Kkh. 23 p. 156.—Cst. Jb. 1852. IV. p. 203.—1853. IV. 167 Anm.—*Wotherspoon*, Cst. Jber. 1844. IV. p. 235.—*Wschiansky*, Cst. Jber. 1862. IV. p. 128.—*Wünstedt*, V.-H. Jber. 1868. II. p. 255.—*Wunderlich*, Arch. f. phys. Hkde. 1858. XVII. p. 15. Eigenwärme in Krankheiten. Leipzig, 1869.—*Young*, V.-H. Jber. 1871. II. p. 249.—*Zavizianos*, V.-H. Jber. 1866. II. p. 246.—*Zehnder*, Schm. Jb. 14 p. 75. Schweiz. Ztschr. f. Hlkde. 1863. II. p. 898.—*Zengerle*, Würt. Corr. 1841. p. 353.—*Ziemssen*, Greifsw. med. Beitr. I. p. 65 d. Ber.

COMPARED with other diseases, the phenomena of which may be of an equally intense and decided character, scarlatina is distinguished by much diversity, not alone in regard to the organs which it affects by preference, but also in regard to the intensity of the disease as manifested in the individual case, in the members of a family, or in the entire epidemic. While in one instance its character is so mild that the patient experiences but little fever, and can move about in the open air without danger, in others, the severest accidents may follow an apparently mild course like this; and, lastly, the disease may be so intense from its very onset that death inevitably results in a few hours. Moreover, during the continued intercourse of people with each other, some individuals will be affected in a mild manner, while others will experience severe and very intense attacks; and lastly, others again, who live under the same conditions, will remain entirely unaffected, though their neighbors may die rapidly or undergo slow consumption with the most alarming symptoms.

The symptomatology of scarlatina is most definitely characterized, first, by a peculiar hyperæmic exanthem of more or less diffusion over the entire surface of the body; and secondly, by the usual occurrence at an early period of an angina of variable intensity. The fever and nervous symptoms are less prominent, but certain phenomena in the joints, serous membranes, kidneys, and subcutaneous cellular tissue are most characteristic. The entire disease is the expression of an infection of the organism by a contagious specific principle.

HISTORY.

In ancient times the character of the exanthem was not deemed of sufficient importance to demand an exact description of it; hence we have no evidence of the prevalence of scarlatina at that period. Unequivocal epidemics of scarlatina, in which sufficient attention was paid to the exanthem, are not found before the sixteenth century. But even in the beginning of the seventeenth century, Sennert refers to scarlatina as only another form of measles; and Morton, at the close of the seventeenth century, in opposition to the then generally accepted differentiation, maintains that both are nevertheless one and the same disease, different only in the character of the exanthem. It is not remarkable, therefore, if the same opinion was occasionally expressed even in the nineteenth century. But Morton's contemporary, Sydenham, established the specific nature of scarlatina by observations made during the epidemics which occurred in London from 1661 to 1675, and thus laid the foundation of our positive knowledge of this disease, which numerous observations of the eighteenth century have materially extended. It was soon recognized that the character of the epidemic was liable to great variation; a series of years during which the disease had been mild and benign were followed by others which manifested an unprecedented malignancy, and established the dangerous nature of the scarlatinous disease. Epidemics of scarlatina have retained this peculiarity up to the latest times, especially in England and Ireland, where, after an unusually mild appearance during the beginning of the nineteenth century, scarlatina, since the fourth decennium, suddenly began to assume not only much more dangerous forms, but also attained a very unusual spread. According to the statistical communications of Farr, the annual mortality from scarlatina in England and Wales from 1848 to 1855 comprised one-twenty-fifth and in some years even one-twentieth of the entire death rate. This also accounts for the many excellent writings on scarlatina which have emanated from British authors. Other civilized states, however, have also suffered much from scarlatina, for everywhere it is a chief factor in mortality statistics. From Europe it has extended over the rest of the world, and the increased commerce of later times has given rise to its appearance everywhere. Thus it has been proved that scarlatina first appeared in Iceland in 1827, in Greenland in 1847 and 1848, gradually extended over large tracts of land in Asia, and lastly, according to Maunsell and Cunningham, also attacked India, which had enjoyed immunity from it for a long time; then it also appeared in Africa, but especially in America (where it appeared first in the North in 1735, but spread very slowly, so that South America experienced its first epidemic in 1829); since 1849 scarlatina has also appeared in Australia. Among modern German authors must be mentioned Hufeland, Kreysig, Stieglitz, Pfeufer, Berndt, Seifert, Göden, Fuchs, Jahn, Eisenmann, v. Ammon, Hauff, Heyfelder, Köstlin, Löschner, Rösch, Schneemann, Kubik, and Steiner; among the French, Rayer, Roger, Guersant and Blache, Barthez and Rilliet, Trousseau and Noirot; among the English, Alison, Bell, Bennet, Brown, Gillespie, Graves, Kennedy, Miller, Murchison, Richardson, and Wood.

ETIOLOGY.

It is indisputable that the *cause* of scarlatina is a peculiar substance which is transferable from the patient to the unaffected individual. Though its existence has been disputed by various authors, their reasons have not deserved earnest consideration. The proofs in support of the contagiousness of scarlatina rest chiefly on the fact that the disease breaks out among a few or many persons in a locality only after the material, which must be looked upon as the cause of the malady, has been introduced into the place—either through the medium of any substance to which it adheres or directly by a scarlatinous patient—and then as a rule only a very short time after the introduction; and further, that these newly affected individuals during their illness likewise produce a substance identical in its properties to the one which originally infected them. But not all individuals in a locality become affected, for the susceptibility is not universal, nor equally developed in those susceptible. When, therefore, scarlatina occurs in a house or family, short intervals elapse between several cases, and the nurses and friends of scarlatinous patients are also much more liable to become infected than others who are perhaps equally susceptible, but remain distant from the morbid atmosphere. It thus happens that numerous cases of scarlatina occur when, in a children's hospital, scarlatinous patients are transferred to the general wards,—an error which was frequently committed in former times. But where the contagious principle has never obtained ingress, there also scarlatina has never made its appearance. The immunity of certain isolated regions, especially islands, from this disease up to the time when the ingress of the contagion gave rise to a diffused epidemic, furnishes proof for this assertion. Other regions became infected only after European commerce had brought over the contagious principle. Isolation of scarlatinous patients, especially in children's hospitals, has likewise prevented the further spread of the disease, and the same result has been attained by the proper disinfection of articles which may have been in the same atmosphere with the patients.

Lastly, we have further evidence of the contagiousness of scarlatina in the communicability of the poison from a patient to a healthy individual by inoculation. This fact, of course, needs further elucidation. Inoculation was first performed with the object of generating a milder form of the disease than the inoculated individual would have experienced naturally. Williams, however, states that this object was not attained; indeed, that the generated disease was as violent as that of spontaneous origin, and that the operation was not repeated after this experience. Rostan speaks of cases in which the scarlet eruption appeared seven days after inoculation. Miquel reported to the French Academy that he had inoculated a number of children, who had never had the disease, with the contents of vesicles from scarlatinous patients; thirty hours after, there appeared at the place of inoculation a red areola, which corresponded in every respect to the scarlatinous eruption; this redness increased for three days, and disappeared on the fifth day; the inflammation was not traumatic, for a second inoculation in the same individual had no effect. It appears, too, that children who had been successfully inoculated were not affected subsequently, when there was abundant opportunity for infection. On the other hand, Leroy denies that the inoculated substance furnishes security; but then he experimented on himself alone, and may not have had any predisposition. According to Guersant, Petit-Radel has ineffectually attempted the inoculation of scarlatina by introducing scales of epidermis under the skin of individuals not previously infected. Stoll, on the other hand, has performed this experiment with success.

Such experiments, in connection with clinical observations, leave little doubt but that the poison is present somewhere in the skin of the patient. It is probably also present in the pulmonary exhalation, for contact with the morbid atmosphere alone suffices to cause infection. Other secretions may also be the carriers of the contagion, as, for instance, the nasal and pharyngeal secretion, perhaps also the urine. The view that the poison circulates in the blood is supported by the fact that children have been born with scarlatina. Moreover, Coze and Feltz introduced a small quantity of scarlatinous blood under the skin of sixty-six rabbits. Of these, sixty-two died in the course of from eighteen hours to fourteen days, having had high temperature, then diarrhœa and emaciation, and the remaining four only recovered after an intense fever. An examination of the blood revealed a peculiar aggregation of the red blood-corpuscles, so that their contour was lost, and the margins of the majority of the isolated corpuscles were indented; in addition, punctate and rod-like, active bodies (bacteria and bacteridia) were found, just as in the blood of the scarlatinous patients who had furnished the inoculated matter. According to Hallier, the blood of scarlatinous patients contains micrococci in great abundance, either single or in colonies, within the blood-corpuscles or on their external surface; occasionally they are also found as short chains and germinating. Riess examined the blood freshly drawn from a vein in the arm of a patient dying of scarlatina, and found that "the serum was filled with an infinite number of small, rapidly oscillating bodies, which, under a magnifying power of five hundred diameters, appeared as dark points between the groups of blood-corpuscles. In addition, there were also rod-like formations, which at many places were recognized as being composed of three or four or more of these first minute bodies disposed in rows." Riess injected a few drops of this blood under the skin of the back of a rabbit, with the effect of developing like small bodies in its blood, and causing its death in twenty-four hours. Further inoculations made with this rabbit's blood gave rise to identical results.

These experiments, therefore, render it highly probable that the contagious principle having entered the blood, is disseminated by it throughout the body, and further, that it stands in some very intimate relation with these finest spores of micrococcus. Hence it is not remarkable that the exhalations from the skin and lungs, and the secretions should contain the contagious principle. The observation of Zengerle, however, that the persons who applied leeches, and who were often much defiled with the blood of scarlatinous patients, did not take the disease, while the mere spectators or visitors became affected with scarlatina, is no argument against the view that the blood is the essential seat of the contagion. For, aside from the fact that only those who are predisposed will become infected, while those who are not can expose themselves without danger, it is certain, as Zülzer has proved, that the contagion of scarlatina can no more enter the organism through the intact epidermis than does the poison of variola.

The shortest contact with the contagious atmosphere of the sick-room may suffice for infection. According to Palante, a mother, after remaining only a moment with a scarlatinous patient, immediately returned home, a distance of about six miles, but communicated the disease to her children, in whom it developed a few days after. Hennig tells of a child who was affected four days after having been but a short time in the company of another child who had been taken sick with scarlatina six weeks before. Köstlin was also convinced that at times a very short contact with a patient was sufficient to cause infection. On the whole, however, the volatility of the contagion of scarlatina seems to be much less than that of measles, and, therefore, spreads less rapidly throughout a dwelling than the latter; on this account the isolation of those in a family who are unaffected, if established at a sufficiently early period and properly carried out, is frequently effectual in preventing a further spread.

It is an undoubted fact that a dilution of the contagious principle by attentive ventilation of the sick-room very much diminishes or entirely removes the opportunity for infection.

The view that scarlatina can be transmitted to unaffected individuals through the medium of substances which have remained in the morbid atmosphere, is indisputably proved by numerous examples. Many cases of so-called spontaneous origin must be explained in this manner.

Thus Richardson attributes the infection and death of a child and its mother to a letter which the former had conveyed to the latter from a house several miles distant, in which scarlatina had prevailed. Petersen attributes the infection of a young girl to the same cause; Maclagan saw a case in which the medium of communication was a woollen shawl, in which a scarlatinous patient had been wrapt; Moore maintains that a piano, which had stood in the sick-room, was the only means of communication. According to Ogle, the contagion which gave rise to a case after the epidemic had subsided, was spread by soiled linen; in another case, two old rock-ing-chairs, with defective cushions, alone could have been the medium. v. Hildenbrand, without the agency of scarlatinous surroundings, became infected by wearing a coat which had been worn formerly upon visits to scarlatinous patients and had been stored away. The first case of scarlatina in the Bahama Islands, that of a child six years old, who had been successfully isolated on the continent, occurred on December 20th, after the patient, according to Duncome, had remained in a region entirely free from the scarlatina poison since October 13th; infection, consequently, was probably caused through the medium of materials which had been brought over from the continent. In one instance Mason Good thinks that a box of toys was the means of transportation. Murchison confidently believes that in a few cases a letter or a lock of hair was the carrier. v. Tscharner saw a case in which the medium was a piece of bedding. In an otherwise obscure case, Behrend's attention was drawn to a bed-pan, which had been used by a scarlatinous patient fourteen months before. Heslop calls attention to the necessity of separating the ordinary washing in a hospital from that which is infected, as he has seen a non-observance of this rule followed by a spread of scarlatina through the whole house, and has also seen it disappear as soon as this rule was obeyed. The spread of the disease, therefore, may be prevented by attentive ventilation and cleansing of all infected substances, especially the clothes and bedding.

Considerable attention was but recently paid in England to the question whether *milk* could become the transporting medium of the scarlatina contagion.

Bell ascertained that several cases of scarlatina had occurred in all houses,—with the single exception of one occupied by old ladies,—to which milk had been conveyed by a peasant and her son, the milk-boy, who had both undergone attacks of scarlatina, and he therefore asks, whether the milk, the receptacle, or the boy was the medium. Taylor observed that one of the first severe cases which initiated an epidemic, occurred in the house of a milkman, whose wife milked the cows; the milk being supplied to about twelve families in the city. In six of these, cases of scarlatina occurred in rapid succession, at a time when the disease was not epidemic, and without any communication having taken place between those that became affected and the person who had brought the milk. It is very probable that in this instance the milk was the carrier of the contagion, as, previous to its distribution to the several consumers, it had stood in a kitchen, which before had been used as a hospital for scarlatinous patients.

It is an indisputable fact that *unaffected* individuals who have nursed scarlatinous patients can spread the contagion, probably through the medium of their clothing.

I observed a case in which a nurse coming directly from a scarlatinous patient communicated the disease, in the short space of three hours, to a child who had almost recovered from a tracheotomy. Willan observed a similar case. According to Williams, Sims relates several cases in which midwives communicated the disease to lying-in women. Many physicians made statements to Murchison, which convinced him that they had transferred the disease by their clothing; this circumstance led obstetricians in active practice to avoid patients with scarlatina. In the above-mentioned case of Palante, a mother communicated the disease to her children, notwithstanding her short sojourn in the infected dwelling and her immediate journey home. Pyle tells of a healthy female teacher, who, without being affected herself, communicated the disease at home by the dress which she had worn while nursing a scarlatinous patient. According to Rehn, a grandmother transported the contagion from Stuttgart to Hanau, thus infecting her grandson. Pons carried the contagion into a small city. According to Köstlin, a child who had been strictly isolated, being convalescent from measles, was taken sick with scarlatina (may the physician have been the carrier ?). Michel saw a family taken sick upon the return of the father from an infected dwelling, after a seven hours' journey. Another physician reports that the contagion was carried five miles by clothing (Arch. d. Vereins f. w. Hkde.). In the epidemic near Eidsvold, in Norway, observed by Thoresen, the intense cold of winter prevented the children from leaving the houses, and the majority of infections (in twenty-four places) could only have taken place through the medium of healthy individuals. Zengerle states that a healthy woman, after a visit to a scarlatinous patient, communicated the disease to her daughter, who was the first patient affected in the whole city.

Just as healthy human beings may be the carriers of the contagion, so also can the poison of scarlatina be communicated to susceptible individuals through the medium of animals. It is also believed that animals may be affected with a disease corresponding to scarlatina in man. Zürn says that Spinola has observed it in the horse; others assert its occurrence in dogs, cats, swine, and other domestic animals. Thus Heim observed that a dog who had lain in the same bed with a scarlatinous child, was taken with fever, followed by scarlatina and desquamation; and it is stated in the Transactions of the Saint Petersburg Medical Society that a cat had a general characteristic eruption, with angina. Letheby speaks of the frequent occurrence of scarlatina in swine, and Krauss relates that during the epidemic of scarlatina at Walddorf, young cattle were affected with cervical enlargements. However, it is still an open question whether the contagion of scarlatina really engenders these diseases in animals, and whether these, in turn, may infect man.

The scarlatina contagion has an extraordinary *tenacity*.

Murchison often found that when a family had left an infected dwelling, fresh cases of scarlatina occurred upon its being reoccupied, after several months; Benedict tells of an instance where several children were affected immediately after their return to a room in which a death from scarlatina had occurred two months before, although it had been thoroughly cleaned and ventilated; Richardson reports that the contagion had become so fixed in the straw roof of a house, that, five months after the first case had occurred, children became infected when they attempted to live in the room under the infected roof; v. Hildenbrand's coat retained its contagiousness for one year and a half. According to Prior, the remaining children of a mother, who had lost two out of three affected with scarlatina, were taken sick after the clothing of one of the deceased was worn, though the residence had been changed; Guersent, Hennig, and Pyle noticed that the contagion retained its integrity for several, Ogle for at least ten, and Fitzpatrick for nine weeks after the disease had subsided in the first child. All these observations indicate that the contagion is not a volatile gas, but a solid material, which easily and firmly adheres to other substances, so that, on account of the resistibility and minuteness of its elements, the ordinary means of purification and cleanliness are inert, and continuous and energetic ventilation can but imperfectly accomplish its removal. According to Hillier, it is only destroyed by heat at the boiling-point; Thoresen states that intense cold does not affect its vitality. In this connection Henry's experiments are noteworthy: he caused unaffected children from six to thirteen years of age to wear the flannel jackets of scarlatinous patients, after these garments had been subjected to a dry heat of 212° Fahr.; notwithstanding the facility with which the contagion is carried by woollen garments, no infection followed. From this we may conclude that it is destroyed by heat, a fact which, according to Behrend, was also proved in Berlin, where the garments were kept in a close and heated chamber, until it had cooled, with the result of destroying the vitality of the poison.

Observations have proved that the duration of the *incubation* (latent period, period of germination) of scarlatina can vary, and certainly is less constant than that of measles and variola. Few but reliable reports show that its period of incubation may be *very short*, differing in this respect from the other two diseases.

The best known case is one which Trousseau relates. An Englishman, travelling with his daughter towards London, from Pau, where scarlatina was not prevalent, arrived in Paris at the same time with another daughter, who, on her way from London, had contracted scarlatina. Both girls lived in the same room, and twenty-four hours after meeting each other, the daughter who had come from Pau was also attacked. In Hanau, where there was no scarlatina, Rehn saw a child attacked two days after its grandmother had returned from nursing a scarlatinous patient in Stuttgart. Russegger saw a child, who had visited a scarlatinous patient at noon, taken sick at night; in another instance, three children visited a sick friend in a neighboring village: two of these children took scarlatina two days after, and the third was affected on the third day. At Wangen, where previously no case of scarlatina had occurred, Zengerle reports that a girl, ten years of age, was taken sick two days after her mother had visited a family sick with scarlatina in a neighboring town. Hennig relates the case of a child, in which the incubation did not extend beyond two to three days at the furthest. Löschner states that a boy, four and a half years old, who entered the hospital for the treatment of a sarcoma, was attacked one and a half days after his admission, and that the hospital could have been the only source of infection. Murchison tells of a lady, twenty-two years old, who was taken sick in less than twenty-four hours from the time at which a visit had communicated the contagion; also of a girl, twenty years old, who was taken sick after a like short period from nursing a scarlatinous patient; of two girls, twenty-one and twelve years old respectively, the former being taken sick after a two, and the latter after a three days' sojourn in an infected dwelling; of a boy, eleven years old, who was taken sick a day and a half after a visit of only two hours' duration to a house in which there were scarlatinous patients; finally, of a man, twenty-two years of age, who became affected three days after a visit to a scarlatinous patient. Voit recalls the case of a child, in whom the incubation lasted three days at most. In a case observed by Marson, to which Murchison refers, the infection must have taken place between twenty-six and a half and thirty-one and a half hours before the beginning of the attack; the stage of incubation could not therefore have exceeded one day and a half at most. Fleischmann observed the infection, by scarlatina, of two variolous children, who had been placed near the scarlatina ward, and in each case three days intervened between their admission to the hospital—*i.e.*, the earliest possible period of infection—and the beginning of the disease. With like certainty, however, the occurrence of a longer period of incubation has been established. Gerhardt reports that a man was attacked with scarlatina four days after an abscess from which he suffered had been opened with a knife used for the same purpose in a scarlatinous patient a few hours before. Murchison saw a girl, four years old, taken sick four days, and a youth, sixteen years old, taken sick four and a half days after having entered an infected locality; also a man, forty-four years old, taken sick five days after visiting an infected locality. Gunz relates a case in which the incubation lasted three days, the infection being traceable to a hospital; the duration was the same in a case of Henoch's, that of a girl who had remained with the infected brother only up to the commencement of the disease, and then had been immediately isolated. In this and similar instances, we cannot, of course, overlook the circumstance that the individual secondarily affected may have been infected at some other time than when the disease commenced in the first patient; for instance, during the period of incubation, if we allow that the contagion may be produced at that time, and then, also, the contagion may have been derived from an older source, such as the clothing or other articles used by the first patient. In this consideration only those cases are of positive value which can be proved to have had but a single contact with the contagion. Pons calculates the period of germination with certainty at four days, in a case to which he had himself brought the contagion; Thoresen, in his above-mentioned observations, estimates it at from two to four days; Zehnder, at from two to five days; Moore reports the case of a woman whose period of incubation lasted seven days, also one of a girl with a period of incubation of not quite five days.

A longer period of incubation was contended for by Veit, who placed it at from twelve to fourteen days; by Paasch, who saw the brother of two infected children taken sick twelve days after they had manifested symptoms of the disease; by Böning, who observed an interval of fourteen days under similar circumstances. Gerhardt was also disposed to accept an interval of from twelve to thirteen days, and assures us that very accurate observations made by his assistant, Reinhold, indicated an interval of eleven days. Now, although the possibility of such a long interval cannot be denied, I am nevertheless inclined to the assumption, on account of the great majority of those cases (occurring in families) which were followed by secondary infections after from four to seven days, that the latter period is the normal, or at least the most frequent interval,—to which view Gerhardt, in his latest publication (Arch. f. klin. Med. XII.) also inclines,—and that, on the other hand, shorter or longer intervals must be looked upon as exceptions to the rule. Some cases, in which the interval is longer, might be classified with those uncertain cases in families where from three to five weeks intervene between the several attacks, and these are certainly not dependent on an uncommonly long stage of incubation, but rather on a retardation of the necessary individual susceptibility; these cases also form the transition to those in which a still longer interval elapses between the affection of different members of a family, and where the action of a new source of infection is necessary to call forth the later attacks. The few successful cases of scarlatinous inoculation would indicate a moderately long duration of the period of incubation; thus that of Rostan, cited by Moore (a supporter of the seven days' view) in which there was an interval of seven days. The difference in the period of incubation of scarlatina from that of varicella was illustrated in a very interesting manner in Hirschsprung's case: a girl, eight years of age, who was ill with both diseases, also infected another girl with both; but in the second girl varicella appeared eleven days subsequently to the scarlatina, which period corresponded to the short incubation of the latter disease, in contrast to the interval of about seventeen days of the former. I think that the latent interval of scarlatina is best ascertained by the observation of such cases as have derived the contagion from a common source, and in which the disease manifests itself simultaneously; this is best accomplished by observing the disease as it occurs in families. In these cases a short interval of from four to seven days generally elapses between the commencement of the affection in the first case,—*i.e.*, probably the earliest period at which the other members can become infected—and the time at which the first symptoms develop in them. Observations like those of Veit and Böning, in which the interval lasted from eleven to sixteen days—the average period therefore comprising about fourteen days—have been reported less frequently. These observations, therefore, which perhaps might be explained as due to variations in the contagiousness of the disease and in the individual susceptibility, cannot alter the rule, that the period of incubation of scarlatina has an average duration of from four to seven days.

In this variation of the incubative period scarlatina differs from all other acute exanthemata, and the cause which occasions it is unknown. It has been asserted, indeed, that it is dependent on the intensity of the epidemic, the severity of the disease, the continued exposure to the contagion, the unfavorable condition of the infected individual; but all these assertions are not sustained by evidence, nor do either measles or small-pox furnish an analogy under similar circumstances. Though it cannot be denied that an intense case may develop a more powerful contagion than a very mild one, I believe that this variation should be ascribed rather to the degree of the individual susceptibility than to the intensity of the contagion; as will be shown later, the individual predisposition to scarlatina is less general and temporarily more variable than in the case of either variola or measles.

The question, at *which period of his affection* a scarlatinous patient may communicate the disease, and the *duration of this capacity of infecting others*, is an important one, and difficult to answer; in the first place, on account of the great difference in individual susceptibility—for only where this exists can we prove the contagiousness of the poison, as long as our chemical and microscopical resources are inadequate to detect it in the secretions and excretions of the patient—and then, because of the above-mentioned extraordinary tenacity of the scarlatina poison and its great power of clinging to objects. The last-named properties will usually render it impossible to determine whether an infection by a scarlatinous patient during the last stages of the disease proceeded from a contagion which was generated at the time of infection, or from one which was produced at an early period of the disease, and which, owing to its tenacity, had retained its vitality up to that time. If we simply follow the facts which are verified daily in regard to the incubation, namely, that single cases in a family, into which scarlatina has been carried by one of its members, as a rule manifest symptoms of the disease a few days after the affection of the first patient, we should incline to the assumption that scarlatina possesses the property of infection from its commencement. This fact is proved by Trousseau's case, if we suppose that the contagion was not carried from London in the garments of the daughter who was first affected, which, of course, is possible though not probable. In like manner, the case of Marson is fairly above suspicion, where the boy who was affected second visited a house in which the first boy had just been taken sick. In this respect scarlatina has an analogy in variola. A patient, in the prodromal stage of variola, sustained a severe injury, and underwent an amputation. Without being aware of the variolous infection, Schaper availed himself of pieces of skin from the amputated limb, for the purpose of transplantation, with the result that the individual on whom the latter operation was performed became infected with small-pox. Now, just as the apparently healthy portion of skin here used contained the variolous contagion, it seems probable that the scarlatinous contagion is also disseminated throughout the tissues of the body in the commencement of the scarlatinous disease, and that they are capable of producing the contagion at that time. On the other hand, however, we must bear in mind that when scarlatina appears in a family and the well are *immediately* separated and isolated from the affected individual, it is often noticed that no other cases occur, though this fact cannot be explained on the ground of an absence of susceptibility to scarlatina, for a fresh opportunity for infection will often call forth the disease in the others. It is difficult to explain this phenomenon other than by the assumption that in the first stages the disease is endowed with only a moderate contagiousness. The view of Girard, therefore, that infection can only take place on the first day of the fever, but not later, is not tenable; for daily experience teaches that scarlatinous patients produce an abundant contagion and may cause frequent infection during the bloom of the eruption, as well as in the later stages of the disease. The contagiousness of the post-exanthematic period is usually ascribed to the scales of epidermis which separate during the process of desquamation; but it seems to me that there is not the shadow of evidence to prove that the contagion is contained in them either exclusively or even chiefly; for it may be presumed that the contagion enters from the blood into all secretions and excretions of the patient. Volz, in fact, totally denies the contagiousness of the epidermal desquamation.

In this connection a report may be of interest, which states that scarlatina occurred in a Berlin infirmary, though the reception of scarlatinous patients was prohibited; dropsical children, however, who had had scarlatina, and, if I remember rightly, had passed through the stage of desquamation, were received into the house without question. In spite of thorough cleansing, etc., the contagion was produced at this late period. Hamburger reports a similar instance: a boy, thirteen years old, was taken sick with scarlatina followed by dropsy, and, three weeks after the commencement of the latter, was taken several miles to his home. Here he remained six weeks, when death ensued. One week before it, his brother, four years old, was taken sick, and soon after other persons in the neighborhood. It thus appears that the first patient was able to develop a contagion in the third month of the disease. Bokai, on the contrary, does not consider dropsical scarlatinous patients infectious, if after a bath they are allowed to remain in the common room.

It is certain that the contagiousness diminishes as health becomes restored; but it is impossible to say when it ceases.

The *individual predisposition* to scarlatina is much more general than that to measles and variola, and in no manner identical with the same. Thus Röser observed that a girl, six years old, did not infect her twin brothers with measles, but that the latter communicated scarlatina to the sister. While almost all individuals are affected by the contagion of measles and variola—in the latter case, where they have not been vaccinated,—many individuals remain exempt from scarlatina, notwithstanding manifold opportunities for infection. This is well illustrated by an observation, among others, of Heyfelder's, who found that during a simultaneous epidemic of measles and scarlatina, many convalescents from scarlatina became affected with measles, while very few convalescents from measles were affected with scarlatina. Epidemics in which, as in the case of measles, the entire population, except those persons who had had the disease before, has been affected (as this occurred in the village of Thalheim (population of 1,300 inhabitants) where all the children and many adults were affected with scarlatina or allied diseases), are exceedingly rare. An intense family predisposition is more frequent, showing itself by numerous and severe attacks of scarlatina among the several members of a family, as soon as one infection has taken place in it; the cause of this is as obscure as the nature of the predisposition. This fact is a sufficient cause of anxiety for the remaining members of a family, when two or more children are attacked in quick succession.

Härlin reports that the parents and nine children of a family were attacked within a short period. Müller reports that in a family of eight children, four out of five attacked died after two or three days. In a family under Löschner's care, all the children, five in number, died within a fortnight; according to Copeman, five children in one family were attacked so suddenly, and four of them with such severity that it was suspected they had been poisoned; these four died and the disease was explained by the regular course of scarlatina in the fifth case. According to Corson, all the members of a wealthy family, composed of the parents, eight children, and two grandchildren, who lived in the healthiest locality, were attacked with scarlatina, and of these twelve individuals, seven children, from one and a half to seventeen years of age, died within six days. Fitzpatrick states that the immediate isolation of four children upon the attack of the fifth child did not save them, for all died within a short time. According to a notice in the Würt. Corresp.-bl., 1857, p. 183, three children were attacked and died within two days after an older sister with scarlatina had been received into the house. In some families Fenini saw the disease manifest an exceptional severity.

Fortunately such an intense family predisposition is exceptional. A greater intensity of the influencing contagion does not explain it, for the infecting cases are frequently of a mild character, and we often find very benign attacks amidst the most malignant, both having evidently originated in the same contagion. On the other hand, there are families in which the susceptibility to scarlatina seems to be entirely absent; such individuals become the carriers of the contagion, without being affected themselves.

When scarlatina enters a household, it is common for only one or several members to become affected, while the others, though not separated from the sick, or at least not isolated with any particular care, remain entirely unaffected. Occasionally this immunity lasts through life in some individuals, in others only for a longer or shorter time.

Thus Moore reports that a woman who had nursed her daughter with great attention, during an attack of malignant scarlatina in 1834, was not then affected, while in 1843 she underwent a very severe attack, having been infected by her two younger children, who had been born since; he also tells of a clergyman who was not infected by his sister who had scarlatina, though he nursed her personally, while one year later he was infected after visiting other patients, and died. v. Hildebrand had certainly attended numerous scarlatinous patients before he took the disease himself. The influence of individuality is so powerful that in a family, children living under the same conditions, having the same parents, and exposed to infection in the same epidemic, may either remain well or become infected in the most variable manner.

When epidemics of scarlatina appear in a population which has enjoyed immunity from it for a long time, the disease does not always find a particularly susceptible population; in this respect scarlatina differs from measles. Thus Hauff, in his description of the epidemic of 1855, states that it was rare for more than one child to be affected in a family, though the disease had been absent for thirteen years. At any rate, it seems as if. at certain times, some individuals are exempt from the predisposition to scarlatina, but that gradually, under the influence of the contagion, it is again developed. It often happens that individuals who have undoubtedly been exposed to the influence of an apparently intense contagion for a long time, are attacked uncommonly long after there is opportunity for infection.

Thus, according to Zehnder's reports, among thirty-two families in which he attended more than one scarlatinous patient, from two to four of the members were attacked at the same time in seven families; in sixteen families, from two to five members, within from five to seven days; in the other families the cases commenced after intervals of one month or even three months. Majer (Ulm) has also observed the latter interval in one family. I observed the case of a boy, who had remained three months in a close room with his brother, while the latter underwent a very severe attack of scarlatina, and who was only attacked with scarlatina upon accompanying his brother to the country, where scarlatina did not prevail, and where they had no intercourse with sick people. Evidently the susceptibility of the second brother was absent during the commencement of the disease in the first, but had developed during the continuance of the disease,—which was marked by dropsy and uræmia—had continually increased, and was finally called into activity by an insignificant circumstance (the contagion in the long-discarded Sunday clothes of the first boy?).

The fact that scarlatinous patients have been transferred to the general hospital wards without detriment to the other patients, must be explained by the limited predisposition to scarlatina, or, more frequently, by its entire absence in many persons; this also accounts for the diminished contagiousness or entire absence of it in the last cases of an epidemic which has appeared in a separate congregation of individuals.

The predisposition to scarlatina seems to be extraordinary in those patients who are affected so intensely that death ensues in a short time. In some cases the continued presence of the contagion may have caused its exaggeration. This intensity is found especially in young children, but occasionally also in adults. Thus, Röser reports the case of a girl, whose sister was undergoing an attack of scarlatina; during the day she vomited several times, but ate with an appetite towards evening; during the night, however, she suddenly died. Such cases are infrequent among adults. In them the predisposition is usually rather slight, and the disease manifests itself generally by the occurrence of a very mild rudimentary affection, even after the individual has been exposed to the influence of an intense contagion for a sufficient length of time.

The question whether *social position* and *external relations*, in other words, nutrition and mode of life, have any influence on the predisposition to scarlatina, has been answered in various ways. Statistics, however, show that the mortality increases with poverty and decreases with affluence (Lievin)—though its dependence on the latter is not so apparent as in the case of measles. But from this the conclusion must not be drawn that position and wealth in themselves are of essential influence; the lesser mortality among the affluent is rather attributable to the better care which their patients receive, to their more complete protection against infection by isolation, and lastly also to the lesser concentration of the poison in their roomier, cleanlier, and better ventilated habitations. If affluence in itself were of any material influence on the individual predisposition, the disproportion between the poor and the rich would be much more manifest than is the case. But scarlatina usually finds its victims in all grades of society, and there is no very marked difference in the number of cases, especially of the severe and rapidly fatal ones, among the several classes of a population. By this fact alone, in the absence of a knowledge of the influencing agents, can we judge of the extent of individual predisposition. The cause, therefore, of the larger number of scarlatinous patients and deaths among the poorer classes, is to be found in the existence of certain conditions which are of minor importance as regards the predisposition in general.

Conditions of the soil may also occasionally exert an influence, especially when some classes of the population are affected either in a very severe or a very mild manner, and when the above law, as an exception, does not seem applicable. It has often been noticed, that while scarlatina attacks one village with severity, a neighboring locality, in spite of the active intercourse between them, remains entirely free from it or suffers very mildly; this fact has frequently been used as an argument against the indisputable contagiousness of scarlatina. We meet with it in the etiology of typhoid fever and cholera, and explain it by conditions of the subsoil essentially independent of any human agency. The same explanation might serve in the case of scarlatina. Thus, if in a large city the wealthier classes live on ground which favors the disease, while the poor live on that which is less favorable, it may happen that the former class will be affected in a greater degree than the latter, while in the opposite case the rich would suffer proportionately less than the poor; it would seem then that the law of an equally divided individual predisposition to scarlatina does not apply in this instance. This exception, however, depends on an accidental, and, as a general rule, rather insignificant complication. Probably every physician can recall instances where numerous and intense affections occurred in families who resided in the most favorable localities. According to Cremen, the wealthy classes of Cork enjoyed an extraordinary immunity in an epidemic of scarlatina; one hundred and twenty-three out of one hundred and twenty-five deaths having occurred among the poor.

The absence of any marked difference between country and city in the number of cases and mortality of scarlatina may be ascribed to the slight influence which external circumstances are capable of exerting on personal predisposition. In contrast to some reports which state that the disease showed a milder character in cities than in the country, as in Baden in 1869, there are others which attribute the greater spread and malignancy of the disease in cities to the overcrowding of many people into a small space. On the other hand, reports of diffused and malignant epidemics in the most isolated localities are sufficiently numerous. In these cases, conditions of the soil probably have an essential influence on the predisposition of the population; further investigations bearing on this question are desirable.

Thus, according to Marchioli, the village of Recorsano, which lies in a swampy region, was alone attacked, and with such severity that forty-four out of five hundred inhabitants were affected, of which number nine died; all the neighboring but better situated villages of the Commune of Vollido remained exempt. Are conditions of the soil responsible for the remarkably intense predisposition in England?

It is possible that a *residence* in *certain localities*, through unknown influences, increases or diminishes the predisposition to scarlatina.

Thus Wood reports that during an epidemic of scarlatina at Edinburgh, out of one hundred and seventy-nine boys in a strictly isolated private institution, forty-four took scarlatina; in another institution containing one hundred girls, not a single case occurred, though outside communication was not prohibited. Richardson described extensive epidemics of scarlatina which occurred among the large and closely crowded crews of the frigates Agamemnon and Odin, and had been occasioned

by the presence of children on these vessels while they were in port; as soon as the frigates gained the high seas, the hygienic condition improved and no further cases occurred. Carpenter finds the cause of an intense scarlatina and the death of all the children on a lonely farm, in the circumstance that blood had been poured on the manure heap which adjoined the dwelling. Cremen asserts that the presence of putrefying substances in the soil, drinking-water, and air augments the predisposition to scarlatina; again, Scott Alison maintains that cleanliness by drainage and good ventilation has no visible influence, to which opinion Hillier also inclines.

Age has a most decided influence on the individual predisposition. While this is very limited in the youngest children, it undoubtedly increases during the second six months, is strongest from the second to the fifth or seventh year, and rapidly diminishes after the tenth year, so that adults, and especially the aged, have only a slight predisposition, the occurrence of the disease among the latter being very rare. Murchison's statistics of 148,829 deaths from scarlatina in England and Wales in 1847, and from 1855 to 1861, show with what frequency the disease occurs at different ages:

	Males.	Females.	Total.
Under 1 year................	5,575	4,424	9,999
From 1 to 2 years..........	10,817	10,158	20,975
" 2 to 3 "	12,324	11,518	23,842
" 3 to 4 "	11,400	11,128	22,528
" 4 to 5 "	9,051	8,675	17,726
" 5 to 10 "	19,219	19,372	38,591
" 10 to 15 "	4,023	4,653	8,676
Under 5 years...............	49,167	45,903	95,070
From 5 to 15 years..........	23,242	24.025	47,267
" 15 to 25 "	1,806	2,065	3,871
" 25 to 35 "	537	769	1,306
" 35 to 45 "	319	352	671
" 45 to 55 "	157	174	331
" 55 to 65 "	89	96	185
" 65 to 75 "	37	51	88
" 75 to 85 "	14	16	30
" 85 to 95 "	3	1	4
Over 95 "	2	4	6
Total.............	75,373	73,456	148,829

According to these, 63.87 per cent. of the deaths from scarlatina occurred under five years, 89.8 per cent. under ten years, 95.63 per cent. under fifteen years of age. On the other hand, only 1.75 per cent. over twenty-five years. It has been asserted that the predisposition to scarlatina is not stronger in childhood than in later years, and that adults only escape the disease because they have experienced it when young. However, if the mortality from scarlatina is calculated at only 6 per cent.—a very low figure—we find, as Murchison says, that the number of individuals attacked with scarlatina in England and Wales is considerably less than one-half the births, so that consequently a large number of people must remain exempt and attain middle age without being protected by a previous attack. Observations of epidemics in isolated regions, where scarlatina seldom prevails, give the same result.

In a Canadian city, according to Stratton, the disease attacked two-thirds of the children, while only five per cent. of the adult population were affected. Panum affirms that, in the last epidemic in Iceland, children and young individuals were almost exclusively attacked, and hence concludes that youth seems to predispose to scarlatina, just as observations on the Faröe Isles have established this fact in regard to measles. Other observers, chiefly German, have obtained the same results. Veit states that only from four to eight per cent. of those affected in Berlin, in 1867, were over fifteen years of age; Gauster treated seventy-eight patients, among whom there were only nine adults; van Holsbeck treated few adults, and none over forty years of age; Bokai seldom saw a patient over nineteen years of age; Berg saw none over twenty-three years old; Belitz, among fifty patients, only had four adults; Böning seldom saw adults affected, and Marchioli never; Fenini saw no cases beyond the age of twenty years; Rösch attended young adults but seldom; Krauss saw few beyond the sixteenth year; among five hundred patients which Vose treated, none were over seven years of age. Zengerle is the only author who saw more adults than children affected during the epidemic near Wangen, in Würtemberg, in 1840. The following authors have observed the disease in older individuals: Braun, in a man said to have been one hundred and forty-one years of age; Ballard, in a patient seventy-four years old; Lees and Tourtual, in one seventy years old; Faber, in one sixty-eight years old; Krauss, in two sixty-two years old, and in four between fifty-three and sixty; Berndt, in a few sixty years old; Belitz, in one fifty-seven years old; Stratton, in a man fifty-one years old. Kelso states that he has seen "old people" affected with scarlatina.

Opposed to these meagre reports concerning the appearance of scarlatina in adults, and especially in older people, there are numerous notices of the occurrence of the disease in children.

According to Fleischmann, they are especially affected between the first and fourth years; according to Bokai, between the second and seventh years; Berg found the predisposition equal in the first, sixth and seventh years, while the ages of one-half of his patients varied between two and five years; Belitz observed that scarlatina generally occurred between the second and eighth years, and Fenini, up to the seventh year; Marchioli never observed it beyond the tenth year; Böning most frequently observed it between the second and ninth years; Rösch observed it up to the sixth year, then the cases gradually decreased until after the tenth year; Röser saw it up to the tenth year; Krauss, up to the eleventh year, and then a rapid decrease; Senfft found that scarlatina occurred especially between the second and ninth years, gradually decreasing up to the fifteenth year; Niese found it especially under ten years; Vose saw it up to the seventh year; Voit, most frequently from the first to the fifth year, less frequently from the sixth to the ninth year, and very seldom at a later period; Löschner found that, except in the first year, it occurred frequently up to the sixth year, when a rapid decrease took place up to the tenth, after which period there were but few patients; Förster saw it from the third to the ninth year, but more especially from the third to the sixth year; Schiefferdecker observed it from the second to the tenth, but more especially in the third, fourth, and fifth years; Gaupp saw it up to the ninth year.

Compared with the numerous observations of the disease during the second and following years, the number of cases which have occurred during the first year are so few in number that we may safely assume for the latter period a very limited predisposition; in this respect scarlatina differs from measles, in which the susceptibility, during the first year, though less, is not so much diminished. It is true that apparently this statement does not correspond with the English statistics given above; we must therefore call attention to the fact, that the number of deaths from scarlatina during the first year does not comprise even one-ninetieth of the entire mortality, whereas the number of deaths from the same cause during the second year represents one-fifteenth of the entire mortality; it is possible that the relative susceptibility during the first year has also increased in England, where the predisposition to scarlatina is stronger.

Haller observed a scarlatinous patient five months old; Fleischmann saw none under six, Eulenburg, none under eight, I, none under five months; Senfft saw only one patient under one year; Gaupp only two; Böning none. According to Bokai,

infants at the breast are rarely affected; the youngest patient that Voit saw was two and a half months old; Küpfer attended a scarlatinous patient two months old. These notices prove that the predisposition to scarlatina during the first year is very slight on the continent.

Nevertheless, even the *youngest individuals* may have a predisposition to scarlatina.

Veit reports the case of a child, born on October 3d, 1856, who was taken sick with scarlatina on the 17th, after the brother, eleven years old, had been seized on the 1st of October. Meynet tells of a woman who, three weeks after the convalescence of her husband, and three months after the death of her child from scarlatina, was delivered of a girl, who was somewhat red when born; the redness became more intense on the following day, and the skin felt very hot. These symptoms diminished after a few days, but again became more intense on the thirteenth day; on the fifteenth day M. noticed the well-developed redness of scarlatina on the whole body, tongue, and palate; the tonsils were swollen, and there was fever and apparent difficulty of deglutition. On the seventeenth day the redness had almost entirely disappeared, and the general condition was good; but during the subsequent fortnight desquamation had not yet appeared, which makes the case somewhat doubtful. Asmus's case is also doubtful, as desquamation frequently appears even in healthy new-born children, in whom there is no suspicion of scarlatina. A mother had lost two children towards the end of her pregnancy from scarlatina, and soon after her confinement became dropsical. In her new-born child, who was very much emaciated, the epidermis over the whole body separated in large sheets; from the hands it came off like gloves; the child afterwards recovered completely. According to Noirot, Potier observed that a woman took scarlatina on the morning of the day of her confinement, and died a fortnight after, and that her child, though immediately isolated after birth, nevertheless became affected with intense scarlatina, but recovered completely.

It is difficult to ascertain whether children are born with scarlatina, because the majority are born with a red or yellowish-red skin, which resembles the eruption of scarlatina, and is followed by desquamation. But as children are born of variolous mothers, or of women who during pregnancy have been exposed to the contagion of small-pox, and at birth have been found affected with the disease, why may not this also happen in the case of scarlatina?

According to Noirot, Baillou, as early as 1574, established the occurrence of such cases, when he wrote: "Uxor Bodini septimestrem partum excussit vi morbi, eodem modo maculatum quo mater. . . . . . Duæ uxores excusserunt partus eodem modo maculatos;" and Ferrario has also published an undoubted case. According to Naumann, F. saw several cases; in one, a dead child, whose skin and digestive mucous membrane were covered with red spots, was born of a scarlatinous mother in the eighth month of pregnancy; from the absence of other signs, the case is certainly a dubious one. Tourtual reports the case of a woman, thirty years old, who had never had scarlatina, and who, after nursing her husband and son—both of whom were sick with the disease—during the eighth month of her pregnancy, up to shortly before her delivery, was delivered of a boy on the 19th of September, 1823; the child could not swallow, and had an uncommon redness of the skin, which Tourtual did not hesitate to consider as a characteristic eruption of scarlatina, the more so as also the tongue and mucous membrane of the mouth were of a glistening red color. The difficulty of deglutition lasted to the fifth day, when the child drank eagerly, and on the ninth day abundant desquamation commenced, just as in older children; later, also, a separation of the nails of the fingers and toes took place; the child recovered completely. During an epidemic of scarlatina, a son was born to Gregory on April 23d, 1839; the child was clearly suffering from some form of fever, and on the following day had a decided angina maligna without an exanthem, as a result of which the child emaciated and died on May 1st. Stiebel tells us that a woman, twenty-five years old, manifested a very abundant eruption of scarlatina toward the end of her pregnancy; after birth, the child could not swallow for several days, the skin was of a scarlet-red color, and desquamated in larger scales than usual.—According to Hüter, a woman, who four days after delivery died of metrophlebitis during an epidemic of puerperal fever, gave birth to a child "præclare scarlatina in stadio florescentiæ affectus."

On the other hand, Murchison has had two opportunities of observing the birth of healthy children by women who at the time were suffering from scarlatina, and Elsässer also reports that a primipara, twenty-six years old, and already affected with the eruption, bore a healthy boy, concerning whose fate nothing is said, so that he probably recovered, while the mother died four days after.

There is no evidence to prove that *sex* influences the predisposition to scarlatina. During the various epidemics it was soon ascertained that while in one case more males were affected, in the other the affection occurred more frequently in females, and generally the difference was inconsiderable. Young boys are more frequently affected, because their number is greater, and women among the adults seem to be more predisposed, because their frequent and close attendance on the sick offers more opportunities for infection.

The assertion by some that *females during menstruation* are predisposed to scarlatina in a greater degree, cannot be sustained by sufficient evidence. The frequent concurrence of the eruption with menstruation proves nothing, for, as in small-pox, the menstrual discharge may be influenced by the disease. In like manner it cannot be said that *pregnancy* favors the occurrence of scarlatina; probably it does not, for Senn, Tourtual, and Trousseau saw no pregnant women affected during severe epidemics; in such cases, according to Miquel, Dance and Hervieux, abortion frequently occurs. *Lying-in women* seem to possess a little more predisposition to scarlatina, as Cremen especially emphasizes; on this point, however, the reports of asylums are unreliable, because a nidus of contagion may possibly have existed in them, and hence new-comers would become affected because, while predisposed, they came in contact with the contagion, and not on account of their pregnancy. This accounts for the occurrence of the disease soon after the delivery. According to the reports of Senn and Hervieux, the disease generally manifested itself after a short period of incubation (from twenty-four to forty-eight hours) in those who were admitted during labor, and it is possible that this short interval is only due to the stronger predisposition of lying-in women to diseases in general, caused perhaps by the exhaustion after labor, the increased irritability, the changed bodily relations, etc. The scarlatina of lying-in women is not to be confounded with the so-called puerperal scarlatina (scarlatina puerperalis in contradistinction to the true scarlatina in puerperal women),—a severe general affection of the system with a scarlatino-erythematous dermatitis, which is called forth by the puerperal state and is not produced by the contagion of scarlatina nor communicable from the patient to other persons, and besides is sufficiently distinguishable by its symptoms from true scarlatina. *Nursing women* possess no greater predisposition to scarlatina, even when their infants are infected, and they have often remained exempt, though not having had the disease before.

Whether any particular *occupation* increases the predisposition to scarlatina is unknown. The majority of those who ascribe their infection to this cause have a right to do so, only because their position in life has brought them into more frequent contact with the poison. Thus, according to Murchison, almost one-third of the scarlatinous patients received into the London Fever Hospital are nurses and servants who have served in families in which the disease has prevailed.

The occurrence of scarlatina in *those who have been operated upon* and in the *wounded* is interesting, and it really seems as if such persons, in consequence of their general condition, possessed a greater susceptibility to the disease. This assumption, perhaps, may not be quite true of the majority of cases in which a scarlatinous eruption appeared under such circumstances; for it must be borne in mind that such exanthemata have appeared under circumstances where there was no suspicion that the poison of scarlatina had been transferred by the operation, or immediately after it, in the hospital. Thus Murchison states that several cases occurred in a London hospital from which scarlatinous patients were excluded with great strictness; May reports the case of a boy, living in a village free from scarlatina, who, six days after receiving a wound on the back of the head, had fever and a scarlatinous eruption, but without angina. But, on the other hand, the fact that the exanthem is said to have been followed by a desquamation characteristic of scarlatina, and also partly by glandular affections, especially cervical suppurations, speaks for its connection with scarlatina. Paget thinks it probable that his patients, who were taken sick a very short time (from three to six days) after the operation, had already been infected with scarlatina before it, but that the effects of the infection would not have set in so soon, and perhaps not at all, if the health had not been disturbed by operative interference. For the present, therefore, we must assume that the etiology of scarlatinous exanthemata after operations and wounds is twofold: besides those cases which very probably or undoubtedly are of scarlatinous origin (and here we must again refer to the great tenacity of the scarlatinous poison and its adherence to substances, instruments for instance) there are others which are less suspicious and can only be classified with the others on account of the similarity of the eruption. The mortality in these cases seems to be greater than in those of simple scarlatina;

Paget believes that many a death occurring a few days after an operation, with obscure symptoms, is attributable to a scarlatina with an anomalous course.

It is doubtful whether previous diseases increase or diminish the susceptibility to the contagion of scarlatina. It is said that scarlatina does not often affect phthisical patients, nor, according to Gillespie, scrofulous subjects; while the same author asserts that deaf-mutes are more susceptible. Children suffering with whooping-cough are also said to escape infection frequently. More extensive experience on these points will only be acquired gradually, and our knowledge will be uncertain so long as we are ignorant in regard to the nature of the predisposition, and can recognize its existence only when the individual becomes affected.

Many individuals possessing the necessary susceptibility have undoubtedly remained unaffected, even after the most intense epidemics of scarlatina; a want of active contact with the contagion, or with a sufficient amount of it, was the only cause of their exemption. Our better knowledge of the same relations in the case of measles proves this to be true.

It cannot at present be decided whether *race* has any influence on the spread and character of scarlatina, as we lack accounts concerning its appearance in distant lands, and as the relations even in our part of the world are in the highest degree variable. Though Hirsch states that all epidemics of scarlatina, which have so far been observed in the tropical and sub-tropical regions, have been of a very malignant type,—in the Antilles, the Brazilian coast, Chili, Peru, Smyrna—these reports have reference chiefly to the white races, which have also experienced epidemics of considerable intensity in Europe. According to D'Alves, scarlatina first appeared among the Brazilian Indians in 1828, and then occurred sporadically; but in the year 1833 and afterwards, it raged with fearful intensity. During the middle of the fifth decennium it was there considered the most malignant disease, and in Montevideo affected whites and blacks in equal degree. According to Murchison, scarlatina has never made a distinction between the several races and the natives of New Zealand; South and North America have suffered like Europe. Stratton, on the contrary, among his Canadian patients had comparatively fewer Indians and half-breeds than French and English; Stevenson, in Pennsylvania, declares that negroes are less susceptible than whites. According to Laudenbach (Majer) Jews are also distinguished by a slighter predisposition to scarlatina, though other authors again deny this.

As a general fact, it cannot be disputed that scarlatina belongs to that class of diseases which occur but *once;* nevertheless exceptions appear to be of comparatively frequent occurrence, as is also the case not only with the other acute exanthemata, but also with the other acute infectious diseases. In the literature, to which I have had access, I have succeeded in collecting, after a rather superficial search—for concerning some cases the notes are incomplete—about two hundred cases of a second infection, besides a few reports of a third and fourth infection in the same individual. It is remarkable, therefore, that Willan, with an experience of two thousand cases, should never have met with a single individual in whom the disease occurred for the second time; while it appears plausible that S. G. Vogel should not have observed a re-infection, as his experience was accumulated in regions where scarlatina seldom appeared. Most writers state that after a close observation of a considerable number of cases during many years, they have never succeeded in establishing the occurrence of a second infection, and that consequently they are compelled to declare the statement of the parents, or of the patient himself, to the effect that he had undergone an attack of scarlatina before, as erroneous; that the previous exanthem was probably due to another cause, and mistaken for that of scarlatina. Now, although as a general rule measles cannot be confounded with scarlatina, the suspicion arises that rubeolæ have often been mistaken for scarlatina, partly on account of the susceptibility of the patients to the disease, which is said to be equally great in some epidemics of scarlatina, but chiefly on account of the similarity of the exanthems and the accompanying symptoms. This may be the reason why for a long series of years, and almost up to a recent period, rubeola has been looked upon as a kind of scarlatina rather than a kind of measles. I believe that my endeavor to understand the diagnostic characteristics of rubeola,—characteristics which may be observed, beyond all doubt, in entire epidemics—has been successful; and I affirm that in the only case of secondary infection of scarlatina and subsequent scarlatinous disease, which I have observed, there was not the slightest possibility of confounding it with measles or true rubeola. This case is the only one among hundreds of cases of scarlatina which I have observed, in which I have been convinced of the possibility of a re-infection; in a few others the statements of the parents were unreliable, and could not be corroborated by the attending physicians. We must therefore assume that secondary infections of scarlatina are of rare occurrence, and this would also appear natural from the fact that the individual predisposition to scarlatina essentially diminishes in the later years of childhood. It is the more remarkable, therefore, that the experience of Trojanowsky in Livonia compels him to almost directly oppose this conclusion, when, after deducting all cases which did not attain full development, he estimates that six per cent. of his cases of scarlatina were recurrences, *i.e.*, secondary infections. Now, if we assume that some of the rudimentary cases must certainly be counted among the recurrences; furthermore, that at the time when he made his report not all of the primary cases were in a condition to be attacked with a recurrence, we must conclude that the susceptibility of the individual to the contagion and its subsequent action is much less often exhausted in Livonia than in other regions. This difference in individual predisposition is probably attributable to conditions of the soil, for Trojanowsky could not ascribe it to age, sex, constitution, hereditary relations, etc.

Scarlatina, as it occurs in this part of the world, is especially distinguished from the majority of the other acute exanthemata and infectious diseases by a peculiarity which is rarely and incompletely present in the case of measles, and is least manifest or entirely absent in the case of variola. This peculiarity lies in the fact that, under the influence of an intense contagion, not only those who have passed the susceptible age without having scarlatina, but also those who have already experienced the disease, are quite often attacked with a usually moderate or mild angina, generally without the slightest trace of a simultaneous affection of the skin, though occasionally the latter shows signs of either an incomplete or fully developed desquamation. Under these circumstances it is evident that even the mildest angina is highly suspicious, and must be ascribed rather to an incomplete infection than to the result of a cold, etc.,—for the differentiation of which, however, our resources are inadequate. Is it possible that in Livonia, under the influence of an unknown agent, these insignificant, more local manifestations may develop into a complete scarlatinous infection? The greater frequency of recurrences in that region might thus be accounted for. To settle this question we require more exact observations regarding the frequency of these mild infections and their connection with scarlatina, and in Livonia a comparison of these similar derangements—if such occur there—with the cases of recurrence.

Putting aside for the present the further discussion of this question, we understand a secondary infection of scarlatina to be one in which the characteristic symptoms of scarlatina are manifested with as much prominence as when we were justified in diagnosticating the first attack of scarlatina. It exists when there is a characteristic exanthem, and may also be assumed when an intense angina is present, due apparently to the repeated influence of an intense scarlatina contagion, but especially when accompanied by a somewhat rudimentary exanthem. In this definition we exclude all those relapses in which the fever and other morbid phenomena again become prominent—though in a less characteristic manner than in the beginning—before the disease has run its complete course. On the other hand, I recognize those cases as new attacks of scarlatina, in which, after a decided retrogression of the symptoms, the exanthem appears a second time in a characteristic manner in company with other morbid phenomena, with or without a subsequent commencement or completion of the desquamation. Those cases only are to be excepted which are characterized by a protracted febrile course, and in which, say during the second or third week, an extensive scarlatinous exanthem, usually transient in character, appears on the whole or greater portion of the body,—an exanthem, moreover, which is more than a simple erythematous hyperæmia. I would designate this incidental phenomenon during the ordinary course as a *pseudo-relapse* or *reversio eruptionis;* those cases characterized by distinct signs of a new scarlatinous infection and following in the wake of the first attack, I would call *relapses;* while, lastly, I would apply the expression, *second attacks of scarlatina*, only to those new cases which occur a shorter or longer time after the first affection, but without any causal connection with it.

Why in rare cases, contrary to the common experience, the susceptibility to fresh infection is not destroyed by one attack is as unexplainable as the causes which usually secure immunity for life after having had the affection once. We can, therefore, only frame hypotheses to explain whether the secondary affection is the consequence of an insufficiently diminished susceptibility, or of one which has increased to an extraordinary degree during convalescence, or of both circumstances combined, especially as we know nothing of the anatomical reasons for the susceptibility. I will, therefore, only mention facts. According to some observers, it seems as if the tendency to repeated attacks were a family peculiarity; thus Robinson states that four members of a family were taken sick simultaneously (without exanthem, however) for the second time. Trojanowsky was able to establish the fact that in two of his cases of secondary scarlatina both parents had also had the disease twice, and in a third case, that at least the father had been affected twice; Murchison observed relapses of scarlatina in two sisters. Sex has no paramount influence. Gillespie alone records the more frequent occurrence of secondary affections among deaf-mutes. In several cases a disturbed development of the exanthem seems to have been of influence, as, according to Trojanowsky, during the first affection it was only developed on the upper, and during the second, only on the lower half of the body, while Berton observed the reverse.

In many of my notes the interval between the first and second affection is stated as having been merely long or short, or that the first affection occurred in childhood, the second in youth or adult age. Older girls and young women appear to be more liable to a second attack than young men,—a fact due, as stated in several reports, to the accidental influence of an intense scarlatina contagion derived from younger members of the family. v.

Pommer's oldest patient (forty-nine years) evidently was infected a second time by his son, who was suffering from a very severe attack. Rudimentary affections of older individuals, during the course of scarlatina in their children, are very common.

In quite a number of cases the interval between the first and second attacks of scarlatina is more accurately stated.

Thus West, after Hillier had already made the same estimate, ascertained it in one case to be thirty-six days, which, though a short interval, is almost too long to allow the occurrence of a relapse in the ordinary sense. Landeutte put it at two months; Holst and Wetzler at three; Salzmann at five; Easton and Trojanowsky at six; the latter at nine; Billing at ten; Cohen and Webster, the latter in two cases, at eleven months; Braun, Henoch, Thomson, Vogt, Wetzler (two cases) and Trojanowsky fixed it at one year, the latter at one and a half years; Salzmann at fourteen months; O'Connor, Trojanowsky (two cases), and Thomas at two years; Salzmann at two and two-thirds years; Salzmann, Thomson, and Trojanowsky, in four cases, at three years; the latter, in two cases, at three and a half years; also in two cases, with Berton, at four years; Trojanowsky at five; Luzsinsky and Clemens at six; Härlin, Trojanowsky, and Höring, in two cases, at seven; Murchison at ten; Lewin at eleven; Clemens at fifteen; Heyfelder, in his own person, at twenty-seven years. Indefinite reports have been made by Bartels, Becker, Cramer, Easton, Elvert, Faber, Formey, Gillespie, Hirschsprung, Höring, Köstlin, v. Pommer, Rupprecht, Seifert, Wood.

As analogous to the relapses of typhoid fever, those cases in which the second attack sets in before the patient has entirely recovered from the first, may be designated as true relapses. But few of the reports inform us regarding the number of days which intervened between the cessation of febrile action and the symptoms of the fresh attack. I do not think, however, that we shall err if we include in this category all those cases of a second attack which are reported as having occurred immediately after the first one, and those also in which it is stated that the fresh affection began not later than four or five weeks after the first. Such true cases of relapse have been described by Bartels, Barthez and Rilliet, Faye, Gaupp, Jenner, Hillier, Kjellberg, Lefevre, Müller, Marshall Hall, Peacock, Richardson, Röbbelen, Schwarz, Smith, Solbrig, Steinbeck, Steinmetz, Steinhal, Stiebel, Trojanowsky, Wood, and an unknown author, according to Thomson. In the majority of cases the first and second attacks were developed with equal completeness; in some cases the second attack was more rudimentary; in a very few cases the first attack was not entirely characteristic. At one time the report states that the first, at another, that the second attack was the severer of the two; in several cases the greater intensity or mildness of the subsequent attack seems to have been determined by the contrary behavior of the antecedent attack, so that both seem to have completed each other.

The relapses among the crew of the frigate Agamemnon occurred somewhat later than usual. According to Richardson, out of about seven hundred men, three hundred were affected the first time, so that the ship had to be cleared, thoroughly disinfected, and ventilated for more than one month. On April 4th it was again manned, and already, on April 9th, relapses occurred in eighteen of the one hundred and two convalescents who had returned to the ship; but the disease also recurred in many of those who had remained on land in the hospital, as also in one who had obtained furlough to return home. It is not reported how many days elapsed between the first and second attacks of the several cases; but as all the first attacks probably occurred in February, and the relapses in April, the interval evidently lasted much beyond one month in most cases. The second affection was either mild or as intense and characteristic as the first.

With the exception of several cases reported by Jenner, Peacock, and Smith, who observed fatal results in children, the relapses of scarlatina always ended in recovery. When the second attack occurs later, it seems that recoveries are still more frequent; at least, I can find but one report of a fatal result, that of West and Hillier, in which case the second attack began thirty-six days after the commencement of the first.

A third and still further attacks of scarlatina are of extremely rare occurrence.

Richardson states that he has experienced scarlatina in his own person three times, and Gillespie has also observed this. Sir Gilbert Blane tells of a young lady who had scarlatina three times, the diagnosis of which was unequivocal. Another case has been related by Bins. Moore reports that a woman, who, when a child, had had scarlatina with angina, and after her first confinement had had angina scarlatinosa without exanthem, was attacked a third time,—three days after a visit to her son, who was sick with scarlatina—and was bedridden for eight days. Thompson reports the case of a girl, sixteen years old, who, three years before, had suffered severe scarlatina at the same time with others of her family, and now had undergone another attack which was followed by dropsy; fourteen days after convalescence from the scarlatina, an affection of the throat set in, followed three days afterwards by a fresh, transient eruption; nine months after this she died from dropsy.

The occurrence of a fourth attack of scarlatina was observed by Stiebel, in a woman about fifty years of age. In four successive years "she had as many attacks of complete scarlatina, the skin desquamating in parchment-like pieces half a foot in length." Stiebel does not state whether the patient had had scarlatina during childhood, or at any other previous time. Jahn relates the following very singular case: A woman, forty-two years of age, who had had scarlatina when six years old, and a recurrence since her menstruation, but without any connection with this function, claimed to have experienced the same disease seven times; she stated that the disease in no respect differed from scarlatina, and in previous attacks had been diagnosticated by eminent physicians as scarlatina; that besides the angina, there was a characteristic exanthem, and after the twelfth day the skin came off first in scales, and afterwards in larger sheets. Jahn calls the disease scarlatina habitualis, attempts to establish its relation with scarlatinoid eruptions, which are due to various causes and circumstances, and adds that in this connection should be mentioned the observation of Henrici, who during the epidemic of scarlatina in Kiel, from 1797 to 1798, attended a woman who was then undergoing her seventeenth attack of scarlatina, with all its symptoms. In the absence of further confirmatory evidence such observations are unreliable.

The *recurrent form* of scarlatina, which Trojanowsky has described, is peculiar. A short time (from seven to seventeen days) after the first eruption of scarlatina, a second eruption, similar to the first, or identical with it, and so to speak completing it, makes its appearance, so that both attacks combined seem to form one complete scarlatinous affection. Both attacks were accompanied by an unusually high fever, similar to that of relapsing fever, and separated from each other, as in the latter disease, by a complete remission, during which all morbid signs disappeared, again to recur with the commencement of the second attack; in addition to the ordinary symptoms of scarlatina, the spleen, as in relapsing fever, increased rapidly in size from the commencement of the attack, the white blood-cells became abnormally numerous, and there was extraordinary prostration together with muscular pains. Now, as most of the patients affected in this manner lived in low and marshy localities, in which relapsing fever occurred almost every year, and also to a certain extent during epidemics of scarlatina, we may freely assume that these cases were the expression of a peculiar combination of relapsing fever and scarlatina. In the meantime we must await confirmations of Trojanowsky's observations.

Cases of scarlet fever occur either in *sporadic* or in *epidemic* form ; it must be remembered, however, that epidemics often continue for so long a time, and the single cases appear to have so little connection with each other, that they might be looked upon as a moderate accumulation of sporadic cases. In the large majority of cases the scarlatina poison can be proved to have emanated from sick persons; or we can, at least, trace its course with more or less certainty from these to the newly affected person. Nevertheless, there are cases which appear to have had no local or temporary connection whatsoever with others, which fact has given rise to the opinion that scarlatina could originate spontaneously through the agency of certain unknown atmospheric and telluric conditions. But if we remember how easily the poison may be transported to great distances, and how long it will in a latent condition adhere to substances, it will be difficult to produce evidence of its ever having originated spontaneously in an isolated instance,—not to mention the question why this individual alone should have been influenced by these hypothetical agencies. There is abundant evidence that scarlatina has never occurred spontaneously in the isolated islands of any latitude, but that it has always been introduced from without by persons or substances. Again, a certain amount of intercourse has always taken place between the most distant localities of civilized countries and the external world. Such cases of apparently spontaneous origin, therefore, only demand a further investigation of the ways and means by which the contagion reaches susceptible individuals. Moreover, the defenders of the theory of spontaneous origin are by no means agreed on the circumstances which control its occurrence. While one attributes it to cold and moisture, others lay it to great heat with moisture, to marshy exhalations, or even to bathing in ordinary river-water, or to any disturbance of the organism itself. We for our part cannot believe that a contagion which determines such positive symptoms can originate in such various ways. When we speak of sporadic cases, therefore, while admitting their origin by means of the ordinary contagion, we mean simply that they have originated without any positive demonstrable connection with other cases of scarlatina, and further, that the cases have not accumulated in sufficient numbers to constitute an epidemic.

The spread of scarlatina in a population which has enjoyed immunity for a long time, and hence probably contains numerous susceptible individuals, often takes place in a manner which, in many respects, seems obscure and inexplicable to us. Some villages which have many cases are located near others in which no cases occur, notwithstanding the active intercourse carried on between the two; while perhaps at a later period, when the disease has ceased in the former village, the latter localities will be attacked in an intense manner. An epidemic spread of the disease from village to village, from district to district, can be established only in exceptional instances. Some districts in larger cities may often have very much scarlatina, while, for some unknown reason, little or none prevails in the others; and then at other times a directly opposite condition may occur without any apparent cause. Though in such cases our attention is directed to the influence which a temporary or local condition may exert in a greater or less degree on the individual predisposition, we must at the same time confess our complete ignorance of the factors which here come into play.

The most complete reports concerning the influence of *seasons* on the prevalence of scarlatina have emanated from England. The greater prevalence of the disease in the fall of the year has been recognized since Sydenham's time; during spring its decrease is not so decided as in other diseases which prevail with like frequency during this time. Of 55,956 deaths in London from scarlatina within twenty-four years (up to 1863 inclusive), 17.87 per cent. occurred during spring, 22.75 per cent. during summer, 35.54 per cent. during the fall, and 23.85 per cent. in winter (the latter comprises the last four months of the old and first nine weeks of the new year); in other words, the largest number of deaths from scarlatina occurred between the middle of September and the middle of November, the smallest number toward the end of March and beginning of April. The greater number of deaths, however, in the fall of the year were certainly not caused by a greater malignancy of the disease at this season. Similar results are obtained by calculating the deaths which have occurred in all England, according to the different seasons of the year.

Other but much smaller data in our own country also indicate the influence of the fall of the year on the spread of scarlatina.

A statement of the number of scarlatinous patients treated by the charity physicians of Leipzig during the years 1842–1869, showed the following results: for the months of December, January, and February, 110, 70, 59 patients; for the spring months, 50, 57, 34; for the summer, 82, 119, 132; but for the fall, the maximum numbers of 151, 161, 144. According to Spiess, during the twelve months of the year the deaths from scarlatina in Frankfort, from 1867 to 1871, amounted to 17, 13; 16, 9, 11; 10, 12, 17; 20, 24, 17; 30. Passow reports that 1,579 deaths from scarlatina in Berlin, from 1863 to 1867, were thus distributed: 101, 68; 91, 89, 113; 131, 151, 105; 155, 228, 179; 168, so that 35.6 per cent. occurred dnring the fall, and 18.6 per cent. of all deaths during the spring months.

Other countries do not furnish such extensive figures as England.

French physicians state (according to Murchison) that epidemics of scarlatina occur more frequently in the spring and summer than at other times. According to Hirsch's statistics, in Scandinavia and Russia, 6 epidemics occurred in the winter months (Dec. till Feb.), 4 in spring, 10 in summer, 13 in the fall; in Germany, the Netherlands, and Switzerland 51 occurred in winter, 38 in spring, 39 in summer, 42 in the fall. Thus in our country a special prevalence of the disease in the fall does not appear to be so marked as in England; but we can only arrive at conclusions by accurate and more extensive estimates, which as yet are wanting. Gutmann's statistics indicate in round numbers about 120 epidemics for the winter, 80 for spring, 90 for summer, 100 for autumn; the tables are compiled from reports of all countries.

The reports of epidemics do not show an aggravation of the disease under the influence of a changeable, cold, and moist *weather*. Of course scarlatina has often prevailed during such weather, but it has not been absent under opposite conditions, and in England especially it has attained a wide spread during warm weather. The condition of the weather can therefore be said to exert but moderate influence on the frequency of scarlatina. It is very probable that its spread is entirely independent of temperature, atmospheric moisture, atmospheric pressure, winds, and electricity. Barker, however, maintains that ozone promotes the frequency of scarlatina, contrary to what is found in other diseases.

Pride states that the condition of the *drinking-water* seemed to be of influence, in so far as the disease appeared more frequently, and with greater malignancy, in those families which used water defiled by organic ingredients, than where this was not the case. But of what disease next, pray, shall we not attribute the cause to drinking-water!

The elevation of the ground has no influence on the spread of scarlatina, for epidemics have occurred in every altitude,—on the sea-coast as well as in the highest localities. Epidemics of scarlatina are likewise independent of the geological structure of the soil; according to Gutmann they occurred on all formations from the primitive rock to the most recent diluvial formations; no difference is occasioned by granite, gneiss, mica, clay, slate, sandstone, shell-limestone, calciferous marl, lias formations, sand, Juracic-, Alpine-, and tertiary lime, tufaceous limestone, turf, or reclaimed land.

On the other hand, *location* and the condition of the *subsoil* seem to have some influence on the spread of scarlatina and the degree of its prevalence.

For this assertion there is abundant evidence. Thus, among other authors, Withering reports concerning the Birmingham epidemic of 1778, that it raged in many high and dry localities, while the inhabitants of moist and low houses were spared entirely or to a great extent. In 1856 Zehnder found that the disease prevailed along the shores of the lake of Zürich, and along the bottoms of the valleys in the higher districts, while in 1857 it prevailed in the higher localities, and in 1858 in the flat lands. Perhaps continued observations concerning the processes which have become so important in the etiology of typhoid fever and cholera, may in future increase our knowledge of the behavior of scarlatina. In this respect we cannot as yet even make conjectures.

Observations on the origin and spread of epidemics of scarlatina continue to prove the paramount importance of *personal intercourse.* Though we cannot understand why this intercourse in a few, or even many cases, has not been followed by a marked spread of the poison, we cannot, at the same time, shut our eyes to the fact that in the majority of cases it is the only causal factor which we are able to demonstrate. In the case of children, the most susceptible class of the population, it is a most important factor, as the contagion is almost sure to spread rapidly through the *schools*, and particularly through the "*Kindergärten*"; the latter have the disadvantage of spreading the contagion among those children who are not yet old enough to attend school, who are at the same time the most susceptible, and in whom the disease is often more dangerous than in school children. In addition, the schools and "Kindergärten" contribute much towards the further spread of the disease, for the reason that the size of the school-rooms does not always correspond to the number of scholars, and their ventilation is necessarily imperfect. If, therefore, the contagion has once entered such localities, it rapidly develops under the favoring influences of warmth, bad air, etc., as in the above-mentioned ships, and the predisposed individuals may thus become infected in much larger numbers than would be possible under the ordinary circumstances of daily intercourse.

But increased personal intercourse cannot always overcome the causes which, in densely populated districts and overcrowded localities, diminish—in some manner unknown to us—the individual predisposition. This explains why smaller localities sometimes suffer more intensely than larger, and why sometimes there is no proportion between the predisposition to and mortality of scarlatina and the density of the population.

Thus, according to Murchison, the number of deaths from scarlatina in four of the poorest and most crowded districts of London, in 1863, was one death in 668 inhabitants, while at the same time, in the four richest districts, the proportion was one in 447 inhabitants.

It is at least doubtful whether scarlatina can be spread in any other way than by either direct or indirect personal intercourse. According to Carpenter the contagion may arise from the offensive odors emanating from the ground, when slaughter-house offal is used for manuring purposes; a number of cases are cited as examples where under these circumstances scarlatina has been of very frequent occurrence. If it is true that animals can be affected with scarlatina, their offal is certainly capable of spreading the disease; but, in accordance with the modern views concerning the nature of the contagion, we must contest the view of Carpenter that it can originate from the blood and tissues of healthy animals. If these substances exert an influence on the spread of scarlatina, it is probably by increasing the predisposition of those living in the vicinity, who become affected with scarlatina in consequence of an accidental contact with the contagion from other regions, not, however, by the spontaneous evolution of the poison from these offensive substances. In support of this latter view it has been found that improvements in privies and sewage in houses have no influence on the spread of scarlatina.

Scarlatina, to a greater degree than perhaps any other disease, appears at one time in the form of a *severe*, at another in the form of a *mild epidemic*, and the same variation is noticed in the sporadic cases, of which many have incorrectly asserted that they are less dangerous than those which occur in the course of an epidemic; Ranke, however, supports the directly opposite view. The epidemic which Sydenham described was so mild that he considered scarlatina hardly deserving the name of a disease, while two years later Morton represents the continuation of this same epidemic as exceedingly severe. Of modern English epidemics the following have been of wide extent and characterized by malignancy: those of 1801–4, 1833–34, 1847–48, 1858–59, but especially that of 1863, which not only raged over the whole country, but also caused so many deaths that the mortality from scarlatina in London during this year comprised one-fourteenth of the entire death rate, or 4,982 : 70,312; in other years it was four times less (Murchison). In Stuttgart during the epidemic of 1846 not one death resulted from scarlatina, so that it may almost be doubted whether the disease prevailed at all at that time. As a counterpart to this we refer to the report of Santlus, who states that in 1840, in Warsaw, 2,559 children died of scarlatina in five months.

That this variable character of the epidemics is the result of local, and not, as was formerly believed, of atmospheric conditions, is proved by the fact that scarlatina may infect neighboring localities in a mild and malignant manner at the same time. Thus Stiebel mentions the epidemic at Frankfort in 1816 as one of the mildest, while the same is described by Kopp in the neighboring town of Hanau as one of the severest.

The slow spread, variable course, and protracted disappearance of the epidemics of scarlatina contrast markedly with the rapid onset, short and high culmination, and rapid disappearance of epidemics of measles. The former never attain such intensity as those of the latter, but, as a rule, hold their once-attained maximum intensity much longer than the latter; or, if they have abated at an early stage, they again rise to the same height after a short time. While it is very common for an epidemic of scarlet fever to be preceded for a long time by sporadic attacks, either single or in small groups, so is it usual for it to be followed by such sporadic cases, which cause the prolonged appearance, often for years and even longer, of scarlatina, even in smaller localities, while in larger localities they give rise to the suspicion that the disease is endemic. This is due to the action of the less volatile, more fixed contagion of scarlatina, which finds susceptible individuals in smaller numbers than does that of measles; its progress is therefore not so stormy as that of the latter, which suddenly attacks a larger number of individuals at one time; on the contrary, it generally spreads from one individual to another in a gradual manner, and therefore requires a longer time to travel through the susceptible classes of the community. This explains why sporadic cases of scarlatina occur more frequently than those of measles, and in regard to the latter the doubt sometimes arises whether we have to deal with a large number of sporadic cases or with a small epidemic; for the same reason scarlatina lacks the regular *periodicity* of measles.

On this last point the opinions of various authors differ, but apparently only to a slight extent, for what one calls a small epidemic, the other declares to be an accumulation of sporadic cases.

Fleischmann maintains that in the case of Vienna the epidemics of scarlatina occurred in systematic succession (1854, 1858, 1862, 1866, 1870), and that the interval between them always amounts to four years, so that he prophecies another for 1874, though the term epidemic for the years 1854 and 1870 (forty-seven and seventy-six cases respectively) is far-fetched. According to Gerhardt, a more or less marked epidemic of scarlatina may be expected about every four or five years. In the case of Dresden, Förster finds irregular intervals between the epidemics: 1824, 1829, 1831–32, 1834, 1839, 1845, 1847, 1851, 1856, 1862, 1867, therefore about every five or six years, with occasional exceptions, when the interval is shorter. Munich, according to Hauner and Ranke, experienced severe epidemics in 1842–43, 1844–45, abundant sporadic cases in 1849-50–51, small epidemics in 1859–60–61 and 1861-62, and towards the end of 1867 only a few sporadic cases; here, therefore, scarlatina is gradually diminishing in importance, as perhaps nowhere else. According to Voit, Würzburg has experienced since 1842 small epidemics only in 1849, 1856, 1863, large epidemics in 1867–68 and 1870–71; according to Küttlinger, Erlangen in forty years has had only two epidemics of considerable magnitude, namely, in 1833–34 and 1840–41–42, while smaller ones were observable in 1826, 1830, 1853–54–55, and in 1857–58. According to Bärensprung and Weineck, scarlatina appeared sporadically in Halle in 1830–31, 1839–1843, 1848, 1849–50, 1857–58, 1859–60, 1868–1871, and epidemics occurred in the years 1832–33, 1844, 1846–47, 1851-52, 1853–54, 1862–63, 1865–66. Of epidemics in Prague, Löschner records those of 1837, 1844–45, 1850, 1853, 1855–56–57, 1860–61; in the interval there were numerous "sporadic" cases. Köstlin states that up to 1866 Stuttgart had epidemics in 1830, 1846, 1853, 1856–57, 1862–63, and since 1849 sporadic cases have often occurred in the intervals. According to Schiefferdecker, severe epidemics occurred in Königsburg in 1859–60–61–62 and 1867–68–69; in 1857 and 1862–65 frequent sporadic cases occurred. Geissler designates the following epidemics for Meerane: 1835–36, 1844–45, 1849–50, 1855–56, 1861, and 1867–68. Leipzig has experienced epidemics of some size since 1842: in 1843–44, 1851–52, 1856–57–58, 1864, 1867–68, 1870–71; sporadic cases, from which the smaller epidemics could not be distinguished, occurred almost every year in about equal numbers. In Berlin, Passow declares the years 1864–65–66, 1866–67, and the latter part of 1867 to be the periods when epidemics prevailed; but scarlatina also occurred outside of these periods. The statistics of Spiess show that in Frankfort-on-the-Main, since 1857, only one considerable epidemic has occurred, namely, in 1861–62–63, and a smaller one in 1868–69; at other times there were more or less numerous sporadic cases.

According to these reports it would seem as if a certain periodicity had shown itself in some localities, while in others it was entirely absent; this periodicity, however, did not show itself with such regularity as in the case of measles. The peculiar behavior of scarlatina is shown, among other circumstances, by the fact that in populous cities, with active external intercourse, quite long intervals occur in which it seems to be entirely absent, as, for instance, in Stuttgart from 1830 to 1846. On the other hand, it is found that in some locations, especially cities, the disease, without being exactly so disseminated that it might be called an epidemic, does not cease to occur, and only varies in frequency at different times. Scarlatina is only endemic in the largest cities, but there it is also influenced by the seasons.

Occasionally scarlatina, like small-pox and measles, assumes a pandemic character, or at least spreads over large tracts of land, as, for instance, according to Hirsch, in Germany in 1818, in Denmark, England, Germany, and France in 1825 and 1826, in Ireland and Russia as well as here in 1832–35, in Germany, Denmark, and England in 1846–49, in the United States in 1821, and over the whole of South America from 1831 to 1837.

The statistics of epidemics compiled by Gutmann show, however, that since the commencement of our knowledge of scarlatina, epidemics have occurred in every year. The factors necessary for its production must therefore be present somewhere at all times.

Some authors recognize a certain relation between the epidemics of scarlatina and those of measles. Thus Löschner states that it is a constant observation to find both the smaller and the larger epidemics of scarlatina following in the wake of epidemics of measles, so that every exacerbation of measles has been followed also by an increase in the number of scarlatinous patients, and that extensive epidemics of scarlatina are also frequently combined with great epidemics of measles. Köstlin makes the same statement. He observed that an epidemic of measles in Stuttgart always immediately preceded a well-marked epidemic of scarlatina, and he is forced to conclude that there is something in the prevalence of measles in epidemic form, which promotes the spread of scarlatina, that possibly the thorough infection of a population with measles disposes it in a peculiar manner for the reception of the contagion of scarlatina. On the other hand, the relation does not appear to be mutual, as great epidemics of measles have not followed scarlatina. In the two epidemics of scarlatina which Ranke observed in Münich, the increase of scarlatinous cases at one time corresponded with the decrease, and at another time with the increase of the cases of measles. But it does not follow that the greater prevalence of measles should always and necessarily give rise to a greater number of cases of scarlatina. Thus scarlatina did not appear in India until a recent period, though measles have frequently occurred. Such facts cast a doubt on the relations of both diseases in the above sense. Gutmann denies that there is any relation between scarlatina and other diseases; that epidemics of scarlatina do not exclude epidemics of other diseases, and are not excluded by others.

It may be said that the *pathogenesis of scarlatina* is still a mystery. The question of how the contagion acts and calls forth symptoms, whether directly by its increase or by the poisonous products which are thereby engendered, or according to the older view, by a species of fermentation, will only be elucidated when its character shall be better understood than at present. Hypotheses appear to me useless. Scarlatina, in this respect, is still the problematic disease which Hufeland (Jahn) spoke of forty years ago.

PATHOLOGY.

Anatomical Changes.

The scarlatina exanthem is an eruption of the skin due essentially to a hyperæmia, and has been variously described by different authors, undoubtedly because its character varied at different times and in different places, in a manner corresponding to the variation of the entire disease.

In the commencement of its development it usually consists of very numerous and closely aggregated points about the size of a pin's head,—larger in some places, but seldom as large as a lentil,—whose redness, not very marked in the beginning, at a later stage becomes quite brilliant. The skin between these points has a natural, pale color; in normal cases these points are equally distributed over the whole surface of the body, with the exception of the face and the palmar and plantar surfaces; on the genitals, especially of the female, they are also commonly less distinct. The scarlet points are most perfectly developed on the neck and trunk, on the thighs, arms, and forearms, while as a rule they are less crowded together and sometimes larger on the legs and dorsal surfaces of the hands and feet than on other parts; in fact they sometimes attain here the size of a lentil, or may even be larger. They are either entirely flat, or, as is more often the case, very slightly elevated, but not near as much so as the roseolæ of measles; the only places where they are more elevated above the surface and resemble more closely the spots of measles are the dorsal surfaces of the hands and feet, and also those parts in the vicinity of the wrist- and ankle-joints, but they are distinguished from the eruption of measles by being usually somewhat smaller, and by having edges which are less indented. The spots are generally nearly circular, though sometimes elongated on the forearms and legs. They are always so closely crowded together that about the same amount of skin is exempt as that which is covered; this circumstance, taken in connection with the tolerably uniform distance between the several points and their nearly equal size, contributes in a great measure to make the eruption a very characteristic one from the very onset. In order to cause a general confluence of the eruption, but little additional growth of the single points is necessary; in severe cases, in which the exanthem is of longer duration, confluence very often occurs, which accounts for the frequent description of scarlatina as a hyperæmia that is uniformly distributed over the whole body. Close examination, however, in most cases of moderate confluence, proves that the redness is not uniform, for here and there on the surface of the body may be observed isolated, pale points of skin, while in cases of greater confluence there may be seen, upon close inspection, pretty well-defined, darker-colored portions, corresponding to the originally isolated red points; only in the case of a strong and vividly red exanthem, do these latter also disappear in the general, uniform, intense redness. A confluence of the originally isolated red points is much more frequent in scarlatina than in measles; in fact it is so frequent that a confluent redness of the skin is often designated as being one of the characteristics of scarlatina; nevertheless, it is easy in the beginning of most cases, by close inspection, to observe its non-existence. In mild cases of scarlatina, with transient, feebly developed exanthem, there is never confluence; the single red points remain isolated up to the time when they become pale and disappear.

Not infrequently, when the exanthem is intense and markedly confluent, there is an increased turgescence of the skin, which then appears stretched and glistening. But we very seldom find œdema of the subcutaneous cellular tissue; perhaps at most there may be a slight puffiness of the eyelids and cheeks, provided these are at all affected.

In my experience the exanthem rarely appears in any other way than has been described above. Now and then in scarlatina, as in all diseases which commence with a violent fever, there is an initial, more or less diffused, not entirely transitory erythema, which may cover the whole body, and which afterwards insensibly merges into the characteristic redness of scarlatina. Then if the redness is moderate, the single points appear as spots of a darker red, and in case of profuse exanthem they disappear in the general intense, scarlet-red injection of the skin. Their absorption in it is the less observable, the more intense and rapidly the exanthem develops.

The description of the exanthem by most authors differs somewhat from the above; the following examples will suffice. According to Barthez and Rilliet, normal scarlatina is characterized by vividly red spots varying in size up to several centimetres; they are roundish and not elevated, and they have uneven and indented margins, which coalesce with their edges, and thus have a tendency to cover the whole surface of the body, but never form semicircular figures like the exanthem of measles; the color is peculiar, owing to the existence of many small, dark-red points on a bright-red base. Where these dark-red points are absent or are not abundant, the color is brighter; where they predominate, it is darker, or of a raspberry color. According to Canstatt, the exanthem consists of many small red points, which soon expand into large, scarlet-red, irregularly margined, erythematous spots, which coalesce and can gradually cover the whole skin with a confluent, lobster-like redness. Neumann has found the skin in scarlatina in a condition of general and intense hyperæmia, and the superficial layers of the cutis at the same time slightly œdematous; the redness begins from numerous small points, which by their coalescence produce a uniform, finely punctated redness of the entire surface of the skin. Niemeyer says that the redness of the skin begins as numerous small, closely aggregated points, which then merge into each other and cause a uniform red surface, while on the other hand we less frequently find the redness of the skin confined to single spots of varying size and irregular form. Hebra describes fine, closely placed points, "which form a flat, apparently uniform redness." G. Simon finds, when the eruption has reached its maximum development, a vivid redness, which forms spots of varying size and irregular form, with intermediate normal skin, or uniformly covers large sections of the body. We thus find that on the one hand too little weight is attached to the originally isolated small red points, which remain isolated throughout the whole course, and may even, when there is confluent redness, stand out as separate spots, while on the other hand an entirely different view of the scarlatina eruption is advanced by the description of large red spots, which can be present at most in a few places immediately before the development of a definite general confluence.

Generally, after the redness of the eruption has increased in intensity up to a certain maximum,—which varies in different cases—it remains stationary a half or an entire day, and then disappears more or less slowly, according to the intensity reached. During the progress of the disease, the color often changes, chiefly in connection with exacerbations and remissions of the fever, but also with the warmer and colder condition of the patient, etc. Thus, for instance, the fact that the eruption becomes pale during the morning might create the belief that its maximum intensity had been passed, while the following evening brings a much more intense, perhaps even the maximum redness; and, *vice versâ*, warmth may for a short time so increase the redness of a faded eruption that an unexpected change for the worse would appear certain, were it not for the absence of other symptoms of a threatening nature and for the transitory character of the increased redness.

It must also be mentioned that the degree of the redness may vary from a pale red to a dark scarlet red; as a rule the redness is in direct proportion to the intensity of the case. A somewhat bluish redness appears when respiration is difficult. In fresh cases and with unimpeded respiration the redness is clear, and disappears completely under pressure of the finger, and reappears when the pressure is removed. If the exanthem has already existed twenty-four hours and longer, the pressed point does not always pale completely, but gives place, especially in very intense exanthems, to a somewhat yellow and perhaps also in some spots violet or bluish coloration, which latter is dependent on the escape from the vessels of the coloring matter of the blood; the redness also is restored somewhat more slowly than in the beginning of the eruption. If the skin is rubbed moderately, or if, as Bouchut did, letters are traced on the skin with a blunt piece of wood, a very interesting phenomenon results: the eruption over the irritated parts immediately pales to a marked degree, thus rendering the writing very prominent for a short time on account of the red surroundings. This depends on an increased irritability of the capillaries of the skin, but is not, as Bouchut asserts, peculiar to scarlatina and to be used as a means for differentiation in doubtful cases. Similar spasmodic contractions of the vessels undoubtedly occasion the peculiar paleness about the mouth in scarlatina; this paleness also abnormally appears in severe cases on other parts of the skin, but is limited to well-defined regions, thus giving a very peculiar appearance to the eruption.

In scarlatina the eruption rarely affects the face in the same degree as in measles, in which disease it is frequently the most intensely affected portion of the whole body. Undoubtedly there are exceptional cases in which the face is as much affected as the neck and trunk, but this is not the rule. Usually the forehead and temporal regions show at first a finely punctate, afterwards confluent exanthem, while the eruption on the nose and cheeks is distinguished by a sometimes diffuse, sometimes circumscribed but tolerably bright congestion; whereas the region of the mouth, and especially the upper and lower lips, the chin and the angles of the mouth, contrast with this by being remarkably pale. This characteristic behavior of the eruption often enables the physician to make his diagnosis at first sight; for the redness in fever is not accompanied, as in scarlatina, by paleness of the mouth, nor is the redness of the cheeks, forehead, and temporal regions as bright as in the latter disease; but much of the redness of the cheeks in scarlatina is attributable to the fever. The ears are often as much congested as the cheeks, but without the characteristic marks of scarlatina; however, a small spotted injection is often found on their posterior surfaces, at least in the beginning of the disease. The hairy scalp usually shows a punctate exanthem, as upon the neck and trunk and upper halves of the extremities. Not infrequently the spaces between the spots are wider on the legs and forearms than on most parts of the body, and sometimes entirely unaffected portions of skin, of small size, are found here, but on the other hand adjoining them there will be portions of equal size, which are uniformly reddened; confluent redness is frequently seen, especially in the vicinity of the joints. The exanthem also extends to the backs of the hands and feet, but the eruption is somewhat different; as in other parts, the roseolæ on the legs are flat, but are frequently papular on the backs of the feet, though between these papules the small punctate spots are not entirely absent; a similar condition is found on the backs of the hands. But the exanthem on the palmar and plantar surfaces is always confluent and only moderately red, even when there is marked redness of the general exanthem; but it is never punctate. On the external genitals of the female the injection is also only moderate; while on the male genitals a punctate exanthem will be found on all the parts except the prepuce and perhaps a portion of the scrotum, which show a general redness. The glans and labia minora may be either very pale or again slightly injected, but not punctate or spotted.

The exanthem quite frequently deviates from the above description; the following variations ought to be mentioned. First, the *partial eruption*. Scarlatina may be developed only on some parts of the body, as, for instance, on the trunk alone, or only on the face (forehead, temples, cheeks), neck, and extremities, or only on a portion of the latter, particularly in the region of the joints; or, again, it may appear only on one extremity or a portion of it, etc. When it appears on the face alone, a correct diagnosis can generally not be made except by weighing the accompanying symptoms; at other times the face remains entirely exempt, while a characteristic eruption covers all other parts. The exanthem, when partial, is frequently anomalous in other respects.

The exanthem may be very unlike the normal eruption of scarlatina, as for instance when it consists in discrete, large *roseo-*

læ. These vary in size from a lentil to a bean or the joint of a finger, are sometimes round, again may have more or less indented margins, are either not at all or only slightly elevated, and certainly not so marked as in measles, and do not have the characteristic central elevations. They are sometimes disseminated over the whole body, including the face, but more frequently are confined merely to some parts, especially the neck and trunk. Sometimes they disappear for a few hours to reappear on the following day for a short time; but they may also persist and gradually develop into a confluent exanthem over the whole or a part of the body. In such cases it is not characterized by dark points, but is rather of a uniformly scarlet color. Undoubtedly the eruption on the face, and the similarity of the roseolæ to those of measles, impart a strange character to this form of scarlatina; nevertheless its nature is easily established by the accompanying symptoms, and especially by the occurrence of confluence. It is said that, notwithstanding the occurrence of the latter, the outlines of the roseolæ may still be distinguished by the existence of darker-colored spots, similar to the dark points which appear in those eruptions of scarlatina which at first are punctate and at a later period become confluent.

According to my experience those exanthems which appear at an abnormally late stage of scarlatina—deserving less significance when they are scanty, but, when more profuse, entitled to the term of pseudo-relapse "or reversio eruptionis"—are specially characterized by the partial, or at least incomplete, character of the eruption, or by its being confluent, with or without the spots spoken of above. Occasionally such an eruption is found in the commencement of the scarlatinous disease, and is then called "scarlatina variegata."

Besides the hyperæmia of the exanthem there is usually only a very slight œdema of the superficial layers of the skin (scarlatina lævigata); but sometimes this œdema is much more considerable, so that one may rightly speak of a slight, general œdema of the skin; and then probably the subcutaneous cellular tissue is also involved in the change to a certain extent. This œdema is found more especially in the face; its occurrence in the lower extremities, which are sometimes swollen in the beginning of the disease, may be due to other causes, such as nervous influences or renal complication. But the swelling of the skin is not always general; it sometimes occurs only in those parts which were characterized by a punctate injection at the outbreak of the eruption; these swellings in the form of small but very abundant papules can perhaps be better felt than seen (scarlatina papulosa). They are found on all parts of the body, and also on the face, where they sometimes cover the forehead in great abundance; most often they occur on the extremities, especially on the legs, the dorsum of the hands and feet, and in the latter situation they may even precede the redness; they are most profuse near those roseolæ which have developed around the opening of a hair follicle, in the regions last-mentioned. A more or less general, lichen-like and very itchy exanthem sometimes occurs in young children.

A miliary eruption (scarlatina miliaris) is frequently engendered by the further development of the papular form of scarlatina. Then miliary vesicles, about as large as a millet seed, and with turbid contents, which are said to have an alkaline reaction, may appear on all parts of the body. They appear chiefly on the trunk, as a result of too great warmth with profuse sweating, or from the irritation of the skin by hydrotherapeutic manipulations; they may appear on those parts of the back which are subject to pressure, as well as on those parts which are the usual seat of intertrigo (inguinal fold, genitals, nape of neck); the miliary eruption is also sometimes found on the face and lower extremities, in the latter case especially on the papules situated upon the legs and feet. In some epidemics miliary vesicles were so often noticed, and in such abundance, that the normal eruption was observed only in the minority of the cases; in other words, the miliary eruption was in fact the characteristic of the epidemic. Such epidemics were formerly called miliary scarlatina epidemics or simply miliary epidemics, and their occurrence was erroneously ascribed to a peculiarity of the scarlatina contagion. Experience, however, has proved that the miliary eruption depends merely on the peculiar disposition of the skin of the patients, and that its contagion can produce the normal scarlatina exanthem in others, just as peculiar circumstances can call forth the miliary form in an ordinary normal case. In ordinary cases of scarlatina the miliary eruption is never so abundant as to obscure the scarlet character of the eruption; usually only a few miliary vesicles can be found on the skin.

We mentioned above that when the eruption is very intense, very minute violet points, due to the escape of the coloring matter of the blood from the vessels, may appear on the skin, wherever the eruption is present. Under the influence of a hemorrhagic diathesis, which may be superinduced by a severe scarlatinous affection, especially under otherwise unfavorable circumstances, *exudations of blood* often take place into the superficial layers of the skin, either in the form of larger points than those above mentioned, or in broad patches of greater or less extent; hemorrhages into the subcutaneous cellular tissue may also occur. According to Hebra, in adults these hemorrhages are found more particularly in the region of the neck or chest, on the back and around the joints; according to Canstatt, more on the inner surface of the upper and lower extremities. In children I have not observed that they occurred by preference in any region; I have observed, however, that various transitions take place to the milder forms of hemorrhage, which are due rather to the intensity of the exanthem. In severe attacks hemorrhages from internal organs and mucous membranes are also of common occurrence. When the small points of hemorrhage are very abundant and diffuse, the exanthem appears, on superficial inspection, as if of long duration. Mild hemorrhages often occur at any period of the disease; they disappear in one place while appearing more prominently at another, etc.; sometimes the miliary vesicles also become hemorrhagic.

Now and then the scarlatina eruption is accompanied or followed by other forms of cutaneous disease, such as herpes labialis, acne, urticaria, pemphigus, ecthyma, varicella-like and pustular eruptions, etc.; there is no connection, however, between them and the scarlatinous exanthem; they belong to the domain of complications and sequelæ. When convalescence begins, and the previously dry skin begins to perspire, sudamina are frequently observed, but they are not to be confounded with the similar appearances caused by the separation of the superficial layers of epidermis, which contain no fluid.

Immediately after the disappearance of the exanthem an abundant formation of boils has also been observed.

In septic conditions gangrene of the skin, with or without gangrene of the subcutaneous cellular tissue, is not infrequently met with. This is found especially on those portions of the skin which have been subjected to pressure (gangrenous decubitus), but also on those portions which were only characterized by an intense exanthem.

Thus Braun, in the epidemic at Fürth, in 1840, saw dark, bluish-red, marbled spots appear, usually first in the pubic region, and from these the epidermis separated, as it does in a blister, and showed the gangrenous and fetid cutis beneath.

Frequently there is no post-mortem appearance of the exanthem in the bodies of patients who have died during the eruptive stage; occasionally, however, when the inflammation has been very severe, the skin still appears red, and in these cases the cutaneous vessels contain much blood. When the latter are injected with a colored fluid, a picture of the exanthem very similar to that in life may sometimes be produced. According to Noirot, the epidermis of scarlatinous cadavers, a few days old, can be more easily removed than from cadavers with a healthy skin, especially from parts of the body which have lain undermost. Löschner ascribes this phenomenon to the effect of a chemically peculiar exudation into the rete Malpighii, whose nature he could not ascertain, and in which, under the microscope, exudation cells of variable number, form, and size could be recognized. Fenwick found the rete mucosum quite thickened, containing numerous round cells with large nuclei. The basement membrane of the sweat glands was also thickened, and the lining epithelium was in some places entirely gone, though in most places it had increased in size to such an extent as to occlude the sweat canals. Some sweat glands contained blood, and were thereby irregularly and very much expanded; in other cases the sweat canals and uppermost layers of the epidermis had a hemorrhagic coloration. At any rate, these changes were all observed in the superficial layers, while the deeper cutaneous layers were normal throughout.

These results might give rise to the supposition that some kind of a relationship existed between the scarlatinous eruption and the sweat glands. In this respect a report of Landenberger is of interest; he observed the exanthem, in a boy ten years of age, on the cicatrix of a burn (covering a space about two feet square), which extended over the thigh, abdomen, and back, "the skin having been destroyed in its whole thickness. The cicatrix was involved in the scarlatinous process, was hyperæmic and œdematous, and a slight desquamation began in it before it commenced in the other parts. This, however, did not end the case, for a portion, about the size of the hand, underwent diphtheritic necrosis, and yet no signs of diphtheria had appeared in the throat or elsewhere. A flat ulcer resulted, with sharply defined, slightly reddened edges, a slimy, grayish, easily bleeding base, and a thin, scanty, sero-purulent secretion. Within a few days the ulcer slowly enlarged, and would have destroyed the whole cicatrix, if death had not occurred from other causes."

The cutaneous affection of scarlatina, which, as we have seen, is not merely a hyperæmia, but is also characterized by an exudation into the rete Malpighii, is not concluded when the characteristic eruption has paled; other disturbances, which have arisen during the inflammation of the superficial cutaneous layers, must also run their course. These result in an excessive production of newly formed epidermis, which is followed, a short time after the exanthem has paled, by an abundant exfoliation. This *desquamation* varies according to the time of its commencement, duration, intensity, and form. It may follow close upon the eruption, or a few days after it, but only in rare cases after the lapse of a few weeks; and the process may either last but a few days, or, when the eruption has been intense and has appeared very early, it may continue for several weeks. It may affect either the whole body—sometimes recurring several times on portions of the body which have been strongly affected—or only certain parts, those for instance which have a thicker epidermis, as the palmar and plantar surfaces. In the latter case it undoubtedly also occurs on the remaining portions of skin, but is not recognizable on account of the minuteness of the scales. To a certain extent the intensity of the desquamation is dependent on the exanthem; an intense exanthem, however, is not inevitably followed by an abundant desquamation, nor a feebly marked exanthem by a slight desquamation. This is proved by the fact, which has been corroborated by many authors, that a characteristic desquamation may occur on portions of the body on which there has been no eruption whatsoever, and also in patients in whom there has been but little redness of the skin, or even none at all. The explanation of this circumstance is, that hyperæmia is not the sole expression of the cutaneous affection, and that increased proliferation of the epidermis can take place without it. The character of the desquamation varies in different patients and on different portions of the body ; it may be branny (desquamatio furfuracea), but is usually lamellar (desquamatio membranacea), and the several lamellæ may be either small and delicate or voluminous and thick. Branny desquamation usually occurs on a delicate epidermis, after a mild exanthem, and usually follows that form in which the punctate portions of skin do not become confluent; it also occurs in the face, excepting the forehead, and greatly resembles the desquamation of measles. The lamellar desquamation is most characteristic on the palmar and plantar surfaces, from which in children broad pieces of dry, unaltered epidermis may be peeled off ; in the case of a finger, for instance, the whole epidermal envelope may sometimes be drawn off like a glove. Broad surfaces on the forehead and abdomen are sometimes found covered with half-dried epidermis only partially attached to the underlying skin. Storch saw portions separate which were seven inches long by three inches in breadth. Sometimes the epidermis is seen to exfoliate in the following manner: it becomes elevated in the form of a small vesicle, about as large as the head of a pin, but empty, and gradually extends towards the periphery, until, by the coalescing of this with neighboring ones, the skin appears to be separated from its connections over quite large tracts. These little vesicles at first resemble the sudamina ; afterwards, besides being dry and containing no fluid, they are distinguished from them by the construction of their base, which consists of completely formed dry epidermis. Such vesicles frequently lose their epidermal covering at an early period, and then appear as circular excavations, which usually extend in a rapid manner by the continued exfoliation of epidermal scales from their margins. The branny and lamellar desquamation on the trunk usually takes place in this way.

Although not the rule, it is yet not a rare thing to find a second desquamation following the first, over a limited extent of skin; for instance, immediately after the eruption has faded, there may be a branny desquamation followed later by one in which the epidermis peels off in lamellæ, or both times the desquamation may be lamellar in character; on the other hand, it is rare for desquamation to occur twice over more extended regions of the body or over the whole of it; a still rarer occurrence is it for desquamation to take place more than twice.

Jadioux describes a case in which a fifth and sixth desquamation appeared within two and a half months after the cessation of the scarlatina, and even then the process did not seem to have ended. In some places the desquamation had a peculiar character; on the scalp it occurred in numerous branny scales, while on the rest of the body it was lamellar in character; on the arms the scales were disposed in parallel circles, like bracelets, several centimetres in breadth, but in such a manner that their free margins were always turned towards the lower portion, and their attached edges towards the upper portion of the limb; on the trunk the desquamation was similar, but its circular form was less manifest.

Several authors have made the observation that the process of desquamation can involve even the nails of the fingers and toes, and that the hair sometimes falls out. Lentin has seen warts drop off after scarlet fever, and Bicker has seen the same thing followed by excoriations.

After desquamation has ceased, the skin usually remains free from further disease, except in cases of relapse, which are followed by renewed and sometimes very complete desquamation. It is very rare, however, for new roseolæ or other exanthems to appear at this time, when the course of the general disease has gone on regularly.

The changes in the *organs of the throat* constitute as essential a feature of the scarlatinous disease as the exanthem itself; the latter may be wanting altogether or may be developed only slightly, while the former will rarely be missed. It is only in exceptional cases that the throat is involved to so slight a degree that one might possibly overlook this feature of the disease.

While it is customary, for the sake of clearness in the description, to recognize different forms of pharyngeal trouble, these are in reality only different grades of one and the same process.

The mildest form of angina scarlatinosa consists in a moderate and uniform redness of the soft palate and neighboring parts, but more especially of the uvula, the anterior palatine arches and the tonsils; in some cases the redness may be confined to only one or more of these parts. The posterior wall of the pharynx usually remains free. Swelling of the mucous membrane and increased secretion are also absent.

When, however, the angina is more intense, the parts involved become swollen, and the redness is of a deeper hue; the congestion also extends, though only to a slight degree, to the anterior half of the velum, as far as to the hard palate, and to the posterior wall of the pharynx, which shows an increased activity of secretion. Upon the reddened mucous membrane, especially of the soft palate, small elevations arise, like the swollen follicles in an ordinary catarrh; they are of a darker red, about the size of a pin's head, and separated from each other by pretty regular intervals; as a rule they progress no further, but sometimes, when the mucous membrane is more acutely inflamed, they burst, leaving behind small, superficial ulcers, which rarely attain the size of a lentil; I have never seen deeper collections of pus resulting from these follicular formations. The œdematous swelling of the mucous membrane is restricted chiefly to the uvula and anterior palatine pillars. The tonsils are also enlarged to a variable degree, and small follicular abscesses sometimes form in them. Generally, in the course of a few days, all these changes disappear entirely.

Where the throat is still more severely affected, the mucous membrane is of a dark, livid color, the parts are more markedly swollen, and there is a more abundant secretion. The redness now involves not only the pharyngeal region, but also the entire mucous membrane of the mouth, even to the lips, and also the nasal mucous membrane; these parts, furthermore, may be swollen, and from the nose there may be an abundant secretion.

The œdema of the soft palate may be so considerable as to render deglutition very difficult, so that when the attempt is made to swallow, the substances taken regurgitate through the nose. The tonsils are usually much swollen, occasionally to such a degree that they touch each other; abscesses in them are more frequent in this than in the previous form. Respiratory derangements, however, do not occur, nor is the larynx, judging from the voice and the entire absence of cough, affected, unless perhaps to a slight degree. The duration of such an intense affection usually comprises two weeks, and enlargement of the tonsils frequently remains after it.

From these remarks it appears that the pharyngitis of scarlatina, in the majority of cases, and at least in those of moderate intensity, is not diffuse and general, but is confined to certain sections of the throat.

For those who wish to satisfy themselves of the specific character of the scarlatinous angina, Härlin recommends the removal of the affected parts from the cadaver. He maintains that the peculiar condition of the throat in scarlatina can be recognized even in those cases in which death has occurred at a late period, and in those which during life showed but slight subjective and objective symptoms. As a characteristic sign he describes a deep, bluish-red injection of the mucous membrane of the tonsils and neighborhood, of the soft palate, of the uvula, of the posterior portion of the tongue, in the neighborhood of the highly swollen papillæ, of the posterior portion of the region of the cricoid cartilage, and of that portion of the pharynx which includes these different parts, and measures about two inches in breadth; he maintains, furthermore, that the peculiarity of this coloring lies in the circumstance that in the direction of its transverse diameter it is very sharply outlined, never passing gradually into that of the neighboring normal tissues; and finally, that wherever the parts are inflamed, the mucous membrane will be slightly swollen, and the submucous connective tissue infiltrated with serum.

The highest grades of scarlatinous angina, besides causing the above changes, may be associated with very marked disease of the connective tissue of the neighboring parts (angina maligna). Besides an intense œdema of the mucous membrane of the throat and parenchymatous inflammation of the tonsils, the region of the parotid and submaxillary glands, and frequently the entire cellular tissue of the neck, may take on such considerable swelling that, in milder cases, large tumors develop at the angle of the jaw, while in intense cases a single broad swelling, hard like a board, involves the whole lower jaw, extending upwards as high as to the temples, and sometimes downwards nearly to the clavicle. In like manner the retro-pharyngeal and laryngeal cellular tissue swells, and then causes respiratory disturbances, similar to those of œdema glottidis, and impossibility of deglutition. Dispersion of these various inflammatory processes may take place, but must be expected in only the relatively mildest cases. Very often extensive abscesses result, either in the tonsils or in the connective tissue. Large cavities filled with pus are thus sometimes found in the tonsils, and they either burst at once or one after the other, leaving indolent ulcers; furthermore, there may be retro-pharyngeal abscesses, abscesses in the neck, etc. Lastly, these inflammations may result in gangrenous destruction. *Gangrene of the tonsils* usually commences at an ulcerated spot, where an abscess has recently broken; sometimes it occurs even before the abscess has discharged, a point on the summit of the inflammatory swelling assuming a livid hue. Gangrene may not only destroy the tonsils so that almost the entire gland comes away in one piece, but the destructive process can also spread in all directions from this point of origin, destroying the arches of the palate, the uvula, and even the whole of the soft palate. Gangrene of the connective tissue of the neck may cause enormous defects; the skin having been destroyed, the muscles are seen discolored and bare, as if dissected out, and the vessels lie exposed to view; not infrequently fatal hemorrhages result from not only the smaller vessels, but also from the carotid and internal jugular. The exposed surface secretes a putrid ichor, and the disease, if it has involved a considerable area and has extended deep, usually ends fatally, with symptoms of general blood-poisoning; recovery can only take place when the gangrenous portions are small, as when the destruction has been confined to the tonsils.

Diphtheria is so often a complication of scarlatinous angina that many authors have assumed that a necessary relation exists between both diseases, and that the diphtheritic inflammation of the throat is a direct effect of the contagion of scarlatina. The fact that scarlatinous diphtheria is not distinguished by any peculiarity of either form, seat, or course from simple diphtheria, proves the incorrectness of this view; like the latter, it may be confined merely to the throat and to parts which are primarily not affected in scarlatina, or can spread to the nose and larynx, or lastly, may affect the latter alone, as also the contiguous portions of the respiratory mucous membrane; it can extend from the throat to the organs of respiration, or from these to the throat; it can remain mild and superficial, or spread in depth and cause ulcerations, perforations of the soft palate, loss of the uvula, destruction of the palatine arches, etc. Diphtheria can be a complication of the mildest, as well as of the severest scarlatinous throat affections; in every case it naturally obscures the characteristic appearance, and augments the intensity and danger of the simple form, partly by the accession of the consequent local symptoms, and partly also by the diphtheritic constitutional infection. It can complicate scarlatina in every stage of the disease; first, in the stage of incubation, so that the symptoms of scarlatina and diphtheria appear simultaneously; or the diphtheritic symptoms may precede those of scarlatina, thus causing the error of supposing that only *one* disease is present; or, as most frequently occurs, after scarlatina has already existed for several days, the throat symptoms suddenly become aggravated by the occurrence of diphtheria, or it appears immediately after the scarlatinous process has run its course in the skin and throat; or it occurs during the convalescence from scarlatina with or without complications. In like manner, scarlatina may at any time complicate a diphtheria, though this is of rarer occurrence than the former case.

In the beginning of the disease the tongue has a more or less extensive white coating, from which the more swollen fungiform papillæ project as red nodules, often surrounded by an additional white margin; at the same time the whole tongue also becomes reddened, especially at the apex and edges. Soon, perhaps already on the second or third day, the entire coating of the tongue together with the superficial epithelial layers, exfoliates either at once or in successive sections in a manner found in no other disease; the tongue then appears of a uniform and usually quite intense redness, without any coating, but with the above-named small nodules; it is also swollen, so that the teeth cause indentures at the margins. Less often the coating is either not cast off at all, or only to a limited extent, the posterior portions of the tongue remaining covered with it, while the anterior half shows the characteristic dark-red coloration and is smooth, except where the papillary swellings project; still less often the swollen papillæ are absent entirely, and then the tongue resembles that of many other diseases. As a rule, the tongue regains its natural appearance during convalescence; as the exanthem pales the redness disappears, the papillæ diminish in size, and the organ again becomes slightly coated. At times an eruption like miliaria appears on the tongue, and is most manifest on its anterior surface. Betz describes this as a clear, serous, or also occasionally sero-purulent exudation upon the surface of the papillæ fungiformes; when the exudation is clear, it appears in the form of dew-drops seated upon these fungiform papillæ. In severe cases, with high fever and well-marked cerebral symptoms, the tongue often becomes dry, and deep and shallow fissures occur in the middle and along the sides, as in other diseases, but at the same time it does not lose its characteristic redness and exfoliated condition. The fissures may be the exciting causes of deeper ulcers and diphtheritic processes.

The *gums* and *mucous membrane of the cheeks* participate in the inflammation only in intense cases, and then are moderately reddened. The mucous membrane of the lips is more frequently and intensely reddened; in severe cases they often swell, become covered with crusts and fissured, and even the seat of deep ulcerations.

When the course of scarlatina is normal, the *mucous membrane of the nose* is usually pale, and its secretion is not increased. When the disease is severe, however, the nasal mucous membrane frequently becomes involved, though probably never primarily; the trouble always originates in a preceding affection of the throat, such as intense inflammation of the mucous membrane, parenchymatous affections of the tonsils (with or without gangrene) or diphtheria of the throat. In the latter case, the mucous membrane of the nose also becomes affected with diphtheria, at least throughout its posterior portions, while those more anterior are frequently only affected with a purulent catarrh; but the formation of diphtheritic membranes may extend even to the nostrils. After the diphtheritic membranes have exfoliated, new ones frequently form. In the other forms of severe inflammation of the throat the nasal mucous membrane, already in the first days of scarlatina, or somewhat later, swells more or less, is reddened, and furnishes a thick or thin purulent or ichorous secretion, which, as in diphtheria of the nose, corrodes the nostrils and upper lip, and causes redness, inflammation, and ulceration in them. This is the much-dreaded scarlatinous coryza. In a favorable case the secretion gradually becomes muco-purulent and then mucous, and at the same time diminishes in quantity; while in an unfavorable case, extensive ulcerations result, especially in the posterior nares, and thus a chronic nasal disease is engendered with a more or less profuse, oftentimes offensive and ichorous secretion; more rarely gangrene of the soft parts and necrosis of the nasal bones result.

The scarlatinous affection of the nasal mucous membrane can also extend to the cavities adjacent to the nose, especially the antrum Highmori.

The *eye* may become affected at different times and in various ways during the scarlatinous disease. In severe cases, and generally upon the appearance of the exanthem, the conjunctiva of the lids and sclerotic shows a glistening appearance with distinct injection of the vessels, and with or without œdema of the lids; this usually disappears sooner or later after the eruption has faded, the process being accompanied by either very little exudation or none at all. In case of diphtheria of the throat, the same process, according to Schröter, also occasionally attacks the conjunctiva of the eye, and often leads to ulcerative destruction of the cornea. Furthermore, according to Schröter, the cornea may also be affected primarily and independently, usually in the way of rapidly progressing abscesses or suppurating ulcers or pernicious kerato-malacia, in which the cornea of one or both eyes, without any marked symptoms, becomes turbid in a few days, is transformed in its totality into a turbid, dirty, grayish-white membrane, and exfoliates piecemeal. Occasionally the inflammatory process may travel from here over the whole uveal tract and cause panophthalmitis and phthisis bulbi. In rare cases a purulent choroiditis or panophthalmitis occurs primarily from embolic processes, as in typhus or puerperal diseases. Lastly, in case of coincident renal disease, a peculiar form of retinitis must be mentioned, which usually appears in both eyes simultaneously and with equal intensity, and which presents the typical picture of retinitis albuminurica upon ophthalmoscopic examination.

During the existence of the scarlatinous eruption, and also immediately after its cessation, affections of the *ear* frequently occur in connection with the throat disease; they are often tedious and may become chronic. Wendt states that while in measles the catarrhal form of inflammation either of the middle ear or of the Eustachian tube is the rule, in scarlatina that form of inflammation is by far the most common which leads to the accumulation of pus or muco-pus in the cavities of the middle ear, and by perforation of the membrana tympani to a discharge of the matter into the external auditory canal (otorrhœa). As a further consequence of the inflammation, important acoustic parts may become bound down by adhesions formed while the mucous membrane was in a swollen condition, or they may even become destroyed altogether; ulcerative destruction of the borders of the perforation in the membrana tympani may also take place, though an extensive acute tissue necrosis rarely occurs in this membrane. It is also rare for the mucous membrane lining the cavities of the middle ear to undergo such tissue necrosis, either with or without an accompanying caries of the subjacent bone; in fact it never occurs, unless the parts have been affected with diphtheria or a very severe inflammation, or the patient has a poor constitution. When the affection is severe the periosteum of the mastoid process, as well as of the squamous and petrous portions, may also participate in the process of swelling and formation of pus; sometimes the bone and periosteum of these portions become diseased, while the auditory apparatus proper remains unaffected. Lastly, we find severe inflammations of the middle ear accompanied by swelling of the skin lining the external auditory canal; also swelling of the soft parts around the ear as collateral or inflammatory œdema; less frequently this œdema is due to the burrowing of matter, especially in the direction of the parotid region.

The mucous membrane of the *larynx* usually remains intact in normal scarlatina; or at most, when the throat affection is intense, it may be affected very mildly. It is only when the angina is very severe, and especially when it becomes gangrenous and is accompanied by inflammation of the connective tissue of the neck—that is, at the commencement of the attack—that the connective tissue at the entrance of the larynx becomes infiltrated, that the mucous membrane in this region becomes ulcerated and the seat of a purulent catarrh, which may last for a longer or shorter period, and may even lead to œdema of the glottis. Œdema of the glottis, however, may appear at a later period of the disease, during the existence of a general dropsy, and either with or without actual inflammation of the laryngeal mucous membrane. The larynx may also become diseased during the course of scarlet fever from still another cause, namely, diphtheria; for the statement that in scarlet fever the diphtheria never extends to the larynx is incorrect. In fact, diphtheria and croup of the larynx may not only occur during the course of an extensive pharyngeal diphtheria, but may even precede it, in some cases manifesting itself as early as during the prodromal stage of scarlet fever.

According to Rühle, diphtheria, when occurring as a complication of scarlatina, sometimes produces very extensive destruction of the tissues. Albers saw two cases in which there were numerous ulcers extending even into the trachea. Franque found the thyroid cartilage entirely destroyed in one case; and in Smith's case the vocal cords and the ventricles of Morgagni were no longer recognizable.

A moderate *catarrh of the bronchial tubes* is quite often present during the early stage of the disease in severe cases of scarlet fever, and when dropsy follows the attack a bronchial catarrh is almost the rule. In those cases where there has been an opportunity of making a post-mortem examination, the reports state that the redness of the trachea and large bronchi was uniform, and that this change, together with a certain degree of swelling, could be followed even into the finest bronchial tubes. In hemorrhagic scarlet fever punctiform extravasations of blood have also been observed throughout the bronchial mucous membrane; and where there has been a diphtheritic complication, false membranes may be found extending a long distance into the lungs.

Pneumonia, either with or without a simple bronchitis, more rarely complicates scarlet fever than measles, and when it does occur it is only in the most severe cases. It develops under a great variety of forms: generally as a lobular broncho-pneumonia, together with bronchitis, though at other times as a lobular or lobar croupous pneumonia. In scarlatina hæmorrhagica extravasations of blood, of varying number and size, may be found in the lungs; where pyæmia follows scarlet fever, wedge-shaped deposits and abscesses may form in these organs. Gangrene of the lung has also been observed, either in connection with or independently of gangrenous affections in other parts of the body.

In one case, where death had occurred as a result of anuria and convulsions, Biermer found that the tissue of the lung, which naturally is peculiarly dry and inelastic, had everywhere become slightly changed: the walls of the alveoli were thicker than usual and beset with numerous nuclei. In the upper lobe of the right lung a hemispherical nodule, about the size of a hazel-nut, pretty tough in consistency, and of a uniform grayish-red color, was found projecting slightly above the surface; this mass, upon more careful examination, proved to be composed of a number of separate small round masses, about as large as millet seeds, which were also to be found singly in the neighboring lung-tissue. The small cells of which these masses were chiefly composed, possessed relatively large and well-defined nuclei, and were so closely crowded together that no trace of the structure of the part could be seen; it was only in the scattered masses that it was possible to determine the fact that the growth of these cells proceeded from the walls of the alveoli. Similar growths were found in the connective tissue of the pleura.

The *lymphatic glands* and the cellular tissue adjacent to them are frequently the seat of disease. As a rule, quite early in scarlet fever the superficial glands (especially those of the axillary, popliteal, and inguinal regions) are appreciably swollen, and a post-mortem examination during the first stage of the disease not unfrequently reveals considerable enlargement of the deeper glands. Most prone to be affected are the lymphatic glands of the neck, owing no doubt to their intimate anatomical connection with the organs of the throat, which fact, together with the skin eruption, should be borne in mind in considering the etiology of the disease. But it must not therefore be supposed that the intensity of the skin or throat affection is in direct proportion to the degree of enlargement of the lymphatic glands, for many cases are met with in which the contrary is true. Frequently a very serious affection of the lymphatic glands is associated with an insignificant angina or with a very slight exanthem, while on the other hand a slight affection of the glands, with a well-marked lesion of the skin and throat, is a much rarer occurrence. Moderate inflammatory enlargement of the glands ends, as a rule, in resolution; while if they are very much inflamed and enlarged, it is almost impossible to prevent suppuration, which event, however, in some cases, takes place very slowly; it will also frequently be found that glands which have at first only been moderately enlarged, and which have remained in that condition for several weeks, will suddenly increase in size and suppurate, sometimes on both sides and again only on one side. Most of these abscesses, after they have been evacuated, heal slowly, but some of them very rapidly; sometimes the pus burrows to a dangerous extent, perhaps as far down as the thorax, and pyæmia may ensue. A still more unfavorable result is where the inflammatory engorgement ends in sloughing, which is most likely to occur when there is a gangrenous or diphtheritic angina. In this case, when the abscess breaks or is opened, the discharge is found to be a poor thin pus, which soon changes to an ichorous fluid; at the same time the opening grows larger and larger, its edges become thinner and keep steadily receding, until finally there is an opening the size of a silver thaler, and, the cellular tissue having been completely destroyed, the denuded muscles and vessels form the bottom of the ulcer. Fatal hemorrhages not unfrequently occur from erosion of the vessels, though blood-poisoning is the most frequent cause of death; now and then, even in this state, the gangrenous process stops, the dead tissues are thrown off, the ulcer becomes a healthy one, and recovery takes place. The gangrenous process may even extend up on to the face (Heyfelder).

As regards the anatomical conditions of the affected cervical glands, Barthez and Rilliet found the submaxillary glands enlarged, hyperæmic, and softened, or, in a later stage, grayish, very compressible, and the seat of considerable purulent infiltration, and in their interior, little abscesses full of thick, creamy pus. The affection of the cellular tissue is undoubtedly of an inflammatory nature, and does not involve the parotid gland; hence the name of parotitis, frequently applied to it, is incorrect. Nevertheless it is very probable that the anatomical changes of the mumps, that is, the isolated affection of the cellular tissue of the parotid region, commonly known by the name of parotitis, more correctly paraparotitis, may occur as a sequel of scarlet fever, independently of the general inflammatory affection of the cervical cellular tissue.

Of quite a different nature are those enormous swellings which extend from the chin back to the mastoid process, and up over the edge of the jaw on to the cheek, and far down on the neck, and are of a reddish or livid blue color, according as their cutaneous covering is more or less stretched and distended; they occur, as a rule, only on one side, rarely on both sides, and when cut into are found to contain nearly pure blood (according to Huber, hæmatoma scarlatinosum). They are probably the product of a hemorrhagic inflammation of the cellular tissue (perhaps in consequence of lymphadenitis), and should by no means be confounded with those effusions of blood due to erosion of large blood-vessels during gangrenous destruction of the connective tissue of the neck.

Extensive infiltration of the cellular tissue, with the formation of abscesses, may occur not only on the neck and in connection with inflammation of the lymphatic glands, but also at all other points and quite independently of the latter.

In one case I observed purulent infiltration of the cellular tissue of the right hand requiring incisions upon the back and palm, and the extension of which only ceased with death; in such cases not unfrequently the periosteum and even the bones become involved and deep destruction occurs.

Severe cellulitis is especially likely to be caused by septic poisoning, as it is seen to occur when absorption takes place from points that are the seat of gangrene; from this, as well as other causes, gangrenous decubitus may obtain in an incredibly short time. Still another cause of cellulitis are the hemorrhages into the cellular tissues during hemorrhagic scarlet fever.

Extravasations may likewise take place in the muscular tissue (Huguenin).

Very often nothing abnormal is found in those cases which die early in scarlet fever with severe brain symptoms; but in many of them there is congestion of the *cranial viscera*, hyperæmia of the brain (studded with red points, and in some portions uniform reddening of the white and darkening of the gray substance) and of the pia mater; the large veins and sinuses are frequently gorged with blood. Other brain anomalies and inflammation of the meninges are not common; meningitis, œdema of the brain, effusion into the ventricles, are observed only exceptionally, while apoplexy, sinus-thrombosis, etc., are exceedingly rare. When, however, after hydrocephalic symptoms, death takes place in the later stages of the disease, œdema of the brain and hydrocephalus will be found, and may be ascribed as the cause of death in those cases in which serious disturbances of the nervous system have been the prominent symptoms. Nothing certain is known concerning those anatomical changes of the spinal cord which the symptoms in a few of the serious cases lead us to suppose may exist.

There is found in scarlet fever no constant or characteristic condition of the *mucous membrane of the organs of digestion*, as was found to be true also of that of the air-passages, and an extension of the throat affection into the œsophagus is as rare as into the larynx, though, according to Canstatt, there is often more or less hyperæmia of the mucous membrane of the former. The mucous membrane of the stomach and intestines, in cases which have been most carefully and microscopically examined, has been found perfectly normal or only affected with some incidental lesion, as for instance, tubercles. A permanent hyperæmia of the mucous membrane does not indicate, as was formerly erroneously thought, an exanthema of the mucous membrane, but denotes in severe cases, as in other acute exanthemata, the presence of a catarrh. In consequence of this, the mucous membrane of the stomach is hyperæmic and swollen, and covered with a tenacious mucus, and there is probably an increased production of epithelial cells both in the interior of the tubuli and upon the free surface of the mucous membrane, and the nuclei of these cells are found in greater abundance than usual in the mucus.

According to Fenwick, in the beginning of scarlet fever the tubuli are filled and distended with a granular and fatty material, or with granules mixed with small cells, which are found in greater numbers than usual in the contents of the stomach, and which, according to Hillier, sometimes retain a tubular shape; after the second or third week, however, the tubuli are less distended, and only their blind extremities are filled with granular material.

Hemorrhagic erosions sometimes occur in the mucous membrane of the stomach. In the small intestine, besides more or less injection of the mucous membrane, as a rule, only at isolated portions of greater or less extent, and in a few cases throughout the whole intestinal tract, from the duodenum to the cæcum and colon, there is a swollen condition of the glands, partly those of Brunner and Lieberkühn, but especially of the solitary follicles and Peyer's patches, presenting a most striking resemblance to the changes found in typhoid fever, particularly if the patches are ulcerated, as was observed by Deiters. The follicular affection, which sometimes causes small extravasations, may, when it is not of an intense degree, exceptionally and at isolated points of the intestine, cause little ulcers, but in scarlet fever the large ulcerations, as seen in typhoid fever, small-pox, or measles, are never met with. Moreover, small extravasations have been found in the mucous membrane, and especially in its villosities, and Fenwick observed one case in which the latter condition was very striking.

Tüngel observed "diphtheritic exudation" on the mucous membrane of the lower portion of the ileum, in a case of scarlet fever, followed by pyæmic symptoms. E. Wagner found the whole thickness of the mucous membrane of the jejunum and ileum, and particularly the villi of the former, filled with cells and granules, but there was no trace of any fibrinous formation; the epithelial cells of the glands were opaque and slightly fatty.

The *mesenteric glands* are generally found hyperæmic, and some of them considerably swollen, and, when cut into, their tissue is found to contain more fluid than usual.

Fenwick has found the *pancreas* in a slightly inflammatory condition; many others have found it quite normal.

The changes in the *liver* consist principally of a more or less albuminoid infiltration of the secreting cells; substantially the same condition in which they are found in many other diseases, simply as the result of fever.

In a very flaccid, exsanguious normal-sized liver of a fatal case, of forty-eight hours' duration, and in the same organ, enlarged by half its size, of a child three years of age, who died at the end of three and a half weeks, of diphtheria complicating scarlet fever, E. Wagner found numerous (here and there one or two upon the surface of an acinus) white granules, for the most part very small, but some of them larger and easily perceptible to the naked eye, which proved to be lymphoid new growths, and beside these he observed, by the aid of the microscope, and particularly in the interacinous connective tissue, numerous collections of cells and nuclei. The capsule of the liver contained similar granules. Huguenin reports the same condition, while Biermer describes a peculiar growth of connective tissue around the liver cells.

Whether such morbid changes are of frequent occurrence is unknown; for generally only the size and degree of congestion of the liver are reported, conditions which may easily vary, and which are sometimes normal, sometimes abnormal; the fact is, no uniform condition of this organ seems to be found in scarlet fever. The lymphatic glands are frequently somewhat hyperæmic, enlarged, and softened. Icterus has several times been observed as a rare complication of the last stages of scarlet fever. According to Harley, the gall is seldom normal, but very poor in its solid constituents, particularly the biliary salts.

There is no uniformity in the morbid anatomy of the *spleen*, as the reports of the different observers vary considerably, many having found this organ of normal size (probably post-mortem?), whether the case died early or late in the disease, others again moderately enlarged, sometimes exsanguious or hyperæmic, of a firm consistence, and sometimes softened. Judging from my own experience, for the most part among cases that have recovered, a slight enlargement of the spleen can, as a rule, be demonstrated in severe cases of scarlet fever, though very frequently, owing to the restlessness of the child, a thoroughly satisfactory examination is not possible.

Wagner found the neoplasms alluded to in the liver also in the spleen, and he describes the texture of that organ as dark red, somewhat softened and studded with numerous exceedingly minute whitish granules, which are distributed along the course of the vessels and, when confluent, form pretty firm homogeneous masses. These new growths were seen very distinctly upon the walls of the medium-sized and minute arteries, as well as upon the corresponding Malpighian bodies, and it is probable that these granules were distributed throughout the whole substance of the spleen. Biermer has observed enormous enlargement of many of the Malpighian follicles. Wedl has found hemorrhages under the capsule of the spleen. The lymphatic glands of the spleen are found in the same condition as those in the liver.

Morbid conditions of the *genitals*, in the early stage of scarlet fever, are most frequently seen in women (Cormack), less often in young girls. The condition most frequently met with is a moderate catarrh of the vagina, with hyperæmia of the mucous membrane without ulceration; among boys, a slight balanitis may occur, with hyperæmia of the urethral mucous membrane. The internal organs of generation in the female are especially liable to be affected during pregnancy, and abortion may take place. Roger found the testes and the tunica albuginea very hyperæmic and somewhat softened, and Weber inflammation of both testes in consequence, he thinks (likewise Barach), of scarlatinous parotitis; in one case it was associated with acute hydrocele. The formation of an abscess is extremely rare, but it has been observed in the mammæ, the labia majora, etc.

Next to the skin and throat, with their pertinent lymphatic glands, the *kidneys* are the organs most frequently affected by scarlet fever. Notwithstanding this, in a great many of the cases which recover, they remain entirely normal, as they have frequently been found in cases which have proved fatal from some other cause, either early or late in the course of the disease. When they are affected there is found, in the first place, a catarrhal condition, characterized by a disturbance of the epithelial elements of the medullary substance, whereby they are thrown off in large masses and washed away (mostly in the early stages of scarlet fever, but exceptionally also met with in the later stages), and secondly an affection of the parenchyma of these organs. In this case the cortical substance is particularly the seat of changes; the morbid process begins in the

Malpighian bodies and then follows the course of the convoluted tubules.[1]

Where there is only a slight parenchymatous inflammation causing no symptoms during life, or perhaps only a slight albuminuria, E. Wagner says that the kidneys are in a condition of congestive hyperæmia either without, or accompanied by, a more or less marked degree of albuminous infiltration. In the severer cases with hæmaturia and albuminuria, the capsules of the kidneys are easily detachable, the organs enormously enlarged (Biermer), and their substance very hyperæmic and softened, and, as a consequence, the seat of numerous capillary hemorrhages. Moreover, at various points the epithelial cells of the uriniferous tubules are found more or less clouded, enlarged, and changed in position, or even entirely destroyed, or frequently, too, pushed off, so that the tubules, which beside them contain white and red blood-corpuscles, as well as various quantities of cylinder-shaped exudation masses, may be completely blocked up, and thus give rise to further disturbances. Circumscribed inflammatory masses are found diffused throughout the stroma, or only at certain points of the same, resembling abscesses and composed of small round cells, the whole presenting an appearance of interstitial or purulent nephritis. Sometimes during scarlet fever regular abscesses form in the kidneys. In a few cases Klebs has found the kidneys hyperæmic, firm in consistence, and not at all or only slightly enlarged, and studded with uniform white points—the Malpighian bodies—which are the seat of nucleolar growths in the connective tissue between the capillary loops, completely compressing the calibre of the vessels and thereby causing anuria. Very exceptionally the severer forms of parenchymatous nephritis are met with, even in the beginning of scarlet fever, but as a rule not before the end of the second or third week. They end in either complete recovery or death, very rarely in chronic disease of the kidneys.

---

[1] According to Taube, in the worst cases the morbid process affects the membrana propria both of the Malpighian bodies and of all the tubuli uriniferi, as well in the cortical as in the medullary substance, in such a way that in it are developed fusiform cells, which enlarge rapidly and completely fill up the interstices between the tubuli uriniferi, but which, like the cells lining these tubuli, undergo generally albuminoid, and if the case be a long one, fatty degeneration. Thus is produced that condition described by Baginsky, who found the external layers of the cortical substance in a state of complete fatty degeneration, and the uriniferous tubes, the Malpighian bodies, and the interstices transformed into a single mass of fat, which even at some points seemed to be formed into drops.

In scarlatina hæmorrhagica, Huguenin has demonstrated a diphtheritic affection of the mucous membrane of the pelvis of the kidney, which had given rise to copious hemorrhage.

Dropsical effusion is a very common result of disease of the kidneys during scarlet fever, and is most commonly seen as anasarca, which, as a rule, is not, but may be, associated with hydrops of one or more of the serous sacs, or with œdema of the lungs or brain, or with a condition of general dropsy. Anasarca is most frequently seen in the face, genitals, and inferior extremities, less frequently in the arms and abdomen, and least often in the neck and heart. In very severe cases every portion of the body is pretty uniformly affected, while in less severe cases it may only be local, and it is not rare to find one-half of the body more affected than the other. The swelling is doughy and shiny, and where the exudation into the subcutaneous tissue is considerable, the impress of the finger remains for some time; occasionally even the muscles are the seat of the œdema. Where anasarca is present, desquamation, as a rule, apparently ceases, probably because of the highly moistened condition of the epidermic scales, and returns only with the disappearance of the fluid. There may be associated with anasarca a moderate degree of ascites, which in severe cases may be excessive and accompanied by hydrothorax and hydropericardium, or by œdema of the lungs and glottis, while hydrocephalus and œdema of the brain may occur without these symptoms or with only a very slight œdema. In the cases of hydrops of the serous sacs, the transuded fluid is, as a rule, perfectly clear, or only very slightly turbid, and when recovery takes place disappears completely.

Genuine inflammations of the *serous membranes*, with seroplastic or purulent exudation, occur now and then; and although they are often observed during the progress of the kidney disease, they are independent of it. Although very rare, meningitis is the most striking of these, covering with its purulent exudation the whole surface of the brain and its sulci, and is, as a rule, associated with a considerable effusion into the ventricles. Or there may be a peritonitis, generally of a moderate type, with a scanty, cloudy exudation, with or without a layer of fibrine. Pleuritis of one side is most frequently met with either quite early or late in the disease, and it may or may not be attended with pneumonia on the same side. The exudation may be pretty clear or clouded, sero-fibrinous, and contain some blood, or it may be pure pus; its quantity may be insignificant or sufficient to fill the pleural sac. The course and duration of this pleuritis are the same as in a primary attack of that disease. Pericarditis is less frequent, and its exudation, for the most part, serous or sero-fibrinous; it occurs sometimes with a pleuritis, and sometimes in consequence of endocarditis, or an affection of the joints.

Inflammation of the synovial membrane of the joints occurs most frequently just when desquamation is commencing, but may occur at any other period. The joint affection is sometimes so slight that externally there are no signs of it, the pain being the only symptom; while again it is of so severe a character that the whole region is very much swollen, and the skin over the joint considerably reddened. It has a serous exudation, and runs, as a rule, an acute course. The smaller joints of the extremities are particularly liable to be affected. Still it is not at all unusual to meet with cases in which the hip, knee, shoulder, and elbow-joints suffer, and now and then the sterno-clavicular, infra-maxillary and vertebral articulations are inflamed. These lesions are all included under the general term scarlatinal rheumatism of the joints. Sometimes, though quite rarely, in place of a more or less intense synovitis acuta with serous effusion, the disease results in suppurative inflammation of the joint, and consequent ostitis and periostitis, with subsequent caries and necrosis; or in fungous periostitis, or in inflammation of the ligaments of the joint, or in relaxation of the joint and spontaneous luxation. As a result of the suppurative inflammation of the joint, fistulous ulcers are formed, the connective tissue between the muscles in the neighborhood of the joints suppurates, the muscles themselves are destroyed, and death ensues from pyæmia.

The *periosteum* and *bone* become diseased, not only in connection with the joint affections or the ulcerations of mucous membranes, as in the nose, throat, and mouth, or in connection with inflammation of the auditory apparatus, but any part may become the starting-point of the disease. The morbid process does not attack the periosteum of all the bones at the same time and to the same degree, but at one point it is more, at another less severe; here it occurs earlier, there later. So also the product of the exudative process varies. It is by no means uncommon for the periostitis to be suppurative and to lead to necrosis; indeed, some have supposed that most of the cases of necrosis occurring in childhood are due to an antecedent scarlet fever.

In some cases of extensive suppurative periostitis, Betz found the ribs diseased in numerous places; Hamburger, the metacarpal and metatarsal bones; Kennedy, separation of epiphyses as a result of scarlatinal ostitis; Graves, inflammation of the cervical vertebræ, and v. Hauff, ostitis at the elbow and knee-joints.

The implication of the *muscles* is shown by the occurrence of isolated abscesses among these structures, or by serous infiltration of single muscles or groups of muscles. But there are other slighter affections, such as pain and difficulty in contraction, and also certain forms of paralysis after scarlet fever, which are of more frequent occurrence than one would suppose from the apparent condition of the parts.

Scarlatinal rheumatism is characterized also by derangements of the *vascular system*. Besides pericarditis, which has already been mentioned, we meet with endocarditis, resulting sometimes in valvular changes and chronic heart disease, sometimes in acute embolic processes. Peri- and endocarditis are sometimes present without a concurrent or a pre-existing affection of the joints. Not unfrequently the cardiac muscle becomes fatty, and its fibres exhibit a peculiar granular opacity, partly as a result of the high fever, and partly on account of other serious derangements. Hurley states that fibrinous coagula in the heart and great vessels are always found at the autopsy. This fact readily explains the presence of emboli in the peripheral arteries. The larger veins often contain thrombi, especially after severe scarlet fever. Stäger mentions the occurrence of thrombosis of the sinuses in scarlatinal diphtheria.

The *blood* is of a dark red color, thin, and generally contains an excess of white blood-corpuscles. The walls of the blood-vessels often imbibe the coloring matter, and thus present the appearance of inflammation.

Different Forms of Scarlet Fever.

One of the most remarkable peculiarities of this disease is the great variety of symptoms which it presents; a variety so great, in fact, that in consequence of the lack of sufficient evidence there is still some doubt whether some of its forms should be included under the general designation of scarlet fever. Formerly all cases of scarlet fever were divided, for the sake of convenience, into groups, according to the general picture of the disease, so to speak; that is, according to the character of the reaction of the organism; hence, besides the usual and the abnormally light forms, a distinction was drawn between erethitic, inflammatory, nervous, and septic scarlet fever. More recently the division of Barthez and Rilliet has been preferred, which distinguishes not only between the primary and secondary symptoms, but also between the characters of the eruption, and thus divides all the cases into those with a normal and those with an abnormal eruption, all other details being referred to the chapter on "Complications." This method has the merit of simplicity, but it necessitates a great many artificial subdivisions of no special value. Not only is it often difficult to say whether the eruption is normal or abnormal, primary or secondary, perhaps, also, whether simple or complicated, for the distinction is not always easy, but it is by no means certain that we do not ascribe too much importance to the affection of the skin. It cannot be denied that the eruption occupies a prominent place among the lesions of scarlet fever, not only on account of its conspicuous appearance and quite regular occurrence, but also because it is one of the earliest signs of the disease, and consequently an important aid in the diagnosis. And yet, we have no right to attach extraordinary significance to the eruption alone, and to regard the changes in all the other organs as complications. Certainly the fact that the affections of the throat, lymphatic glands, etc., sometimes occur as a result of infection by themselves without a trace of scarlatinal eruption, shows conclusively that they have essentially the same pathological significance as the cutaneous lesion.

A clear view of the disease is possible only when we know the fundamental anatomical conditions, and not only those of a general character, but also those which underlie the affections of particular organs. Other principles of distinction, such as the peculiar disposition of the individual, the composition of the blood, etc., may perhaps satisfy the demands of science better than a purely anatomical principle, but they have as yet been so little investigated that they are not available as guides in classifying the numerous varieties of the disease.

Besides the classes of cases characterized by marked local lesions, there are other cases, in which no local symptoms are present, but simply a fever of greater or less severity, sometimes lasting only a few hours. When the fever is intense, death speedily follows; in other cases recovery; and the symptoms can be best explained by a *poisoning of the blood.* As a subdivision of this, which is the most common form of scarlet fever, we have those cases, in which, besides symptoms of blood poisoning, there is also a hemorrhagic diathesis. It is probable, but yet not satisfactorily proved by observation, that symptoms of severe infection may occur and rapidly cause death, with signs of primary collapse, and yet without any fever.

Finally, as forms transitional to the groups about to be described, we occasionally have cases in which the scarlatinal poison excites fever, which is for some time the only symptom, but is afterwards, either during its course, or after its cessation, followed by local lesions.

The existence of *local affections* gives rise to two further classes, viz.: (1.) those in which the local affections are *single* or isolated, and (2.) those in which they are *numerous* or combined.

In the first of these classes are included cases in which, under the influence of the scarlatinal poison, a single organ is affected, either with fever, or exceptionally without—the local disease being the only sign of the infection. The parts most frequently affected are the skin and the mucous membrane of the throat, the lesions of which are characteristic of the disease. In rarer instances the parenchyma of the kidneys, or the lymphatic glands especially of the neck, or the connective tissue of the neck, may each become diseased as a primary and isolated affection. The same thing is true of the parotid gland, the joints, perhaps also some of the serous membranes, and the mucous membrane of the alimentary canal. v. Ammon states that he has seen abscesses produced by scarlatinal poisoning without the occurrence of disease in any other part.

To group together into distinct classes those cases in which several organs are affected is by no means an easy task, because it is not yet satisfactorily settled which lesions should be regarded as the original localizations of the scarlatinal poison, and which as merely complications. If no distinction were made, and I admit that such a view has much in its favor, the number of separate groups would be very large, on account of the number of combinations possible. Until this question is settled, it is better to consider as original localizations only those forms of disease which are known to occur by themselves when the patient has been exposed to the scarlatinal poison; as, for instance, the above-mentioned lesions of the skin, throat, cervical lymphatic glands, kidneys, also scarlatinal rheumatism, and perhaps also the so-called parotitis. The lesions of all the other organs are complications.

There is still another series of groups, which for the sake of clearness deserve to be distinguished from each other. The basis of distinction is the nature of the individual lesions, and whether they occur together or succeed each other. The line of demarcation cannot be sharply drawn, because in a given case the severity of the diseases of the different organs is very variable.

The following forms of scarlet fever are those which deserve special notice:

The usual forms, characterized by the simultaneous affection of the skin and throat, which may be the only symptoms, or may be accompanied by disease of the cervical lymphatic glands, or of some of the joints, or of the kidneys; in fact, by disease of any one of these parts alone or in succession, and generally in the above order;

The cases of scarlatina without eruption. These attacks, it should be noted, are sometimes followed by a more or less well-marked and extensive desquamation. The throat affection may be very light or very severe, and may be accompanied or followed by a great variety of affections of the lymphatic glands, cervical connective tissue, joints, and kidneys;

The rare cases of scarlatina without angina, and yet with rheumatism, kidney disease, parotitis, and infiltration of the cervical connective tissue in every possible order of succession;

The cases of nephritis, or rheumatism, or parotitis, or disease of the cervical connective tissue, with or without angina, and with a subsequent eruption in all its degrees of development;

The cases of opposite combinations of the above-mentioned local affections without eruption or angina; *e.g.*, disease of the lymphatic glands, and subsequent nephritis (Hamburger); rheumatism, and subsequent disease of the lymphatic glands, followed later by nephritis (Zehnder); dropsy, and afterwards disease of the lymphatic glands (Hamburger), etc.

If now we consider that all these different varieties may be combined in the most complicated manner, and may appear early or late, as severe or as trivial affections, we can easily understand that the scarlatinal poison in its action upon the body, may produce a disease of an extraordinarily varied character. In order to give an accurate description of the rarer forms, it would be necessary to enter more fully into details and nice distinctions than would be justifiable in an article of this kind, and I shall therefore refer to them only in a general way.

The Course of the Disease in its Usual Forms.

After a period of incubation, which is usually entirely latent, but sometimes presents transitory symptoms of slight importance, scarlet fever begins, in normal cases, with fever, followed very soon by cerebral and digestive disturbances, and also by pain in the throat. In many cases the initial fever is not to be distinguished from that of other diseases, but generally it may be recognized by its unusual intensity. Not unfrequently there is more or less prostration; in cases of moderate severity the extremities, and in fact the whole skin, become cold, and in severe cases a rigor of variable duration occurs. In children convulsions often occur. Then follow immediately excessive exhaustion and heaviness in the limbs, with extreme malaise and a considerable elevation of temperature, which in a child, who but a few hours previously was entirely healthy, may reach 104° and upwards, together with other symptoms of intense fever. The cerebral symptoms which occur at the outset of the disease are correspondingly severe; sometimes, however, of little importance. There is severe headache, at least in the older children, with giddiness and dulness of the mind, sleeplessness, or drowsiness, or restless sleep. Urgent vomiting with exhaustion and convulsions occurs more frequently as an initial symptom of this disease than of any other in childhood, except small-pox and pneumonia. It often makes its appearance quite unexpectedly as an initial symptom without other evidence of disease. In mild cases the symptom is generally not of an urgent character. Diarrhœa sometimes occurs, but usually it is not troublesome. The tongue is thickly coated, and the appetite entirely lost. All of these symptoms, however, attract but little attention in comparison with the fever, when it has been rapidly developed.

The throat symptoms are very characteristic. Many patients, especially older children and adults, complain of pain in swallowing, or at least of a pricking sensation in the region of the tonsils as the first symptom. Soon after this the tonsils, uvula, and soft palate become reddened. But there are cases in which the angina is entirely absent, or is developed subsequently; and, on the other hand, the same symptoms may be produced by simple tonsillitis without scarlet fever.

All the other morbid phenomena which occur at this time, such as pain in various parts, especially in the loins, or as symptoms on the part of other mucous membranes, etc., are either of a quite subordinate character or belong not to normal scarlatina but to the beginning of irregular varieties of the disease, or to complications.

With the chill, which in older children and adults, occasionally lasts for some time, and with the more or less complete and serious attack of convulsions in the younger children, the temperature, at the very outset of scarlet fever, generally rises rapidly to a considerable height; in rarer instances it rises slowly with progressive exacerbations and remissions, or remains at a low point. This intense beginning of the disease characterizes the normal cases. A different course of the initial stage does not exclude a normal course, but is often an early indication of the commencement of an abnormal development of the disease. On the evening of the first day the temperature often reaches the height of 104° to 105.8°; the pulse increases in frequency correspondingly, but sometimes becomes disproportionately rapid, reaching 160 beats per minute, or more; there is urgent thirst; the skin is dry and hot; the eyes congested; the malaise most distressing. By this time there are often marked cerebral symptoms, such as restlessness, severe headache, somnolency, screaming, tossing about in the bed, talking in sleep, and delirium.

In the meantime, even at this early period, the local symptoms have begun to assume prominence. Not only are the throat symptoms more severe, as is shown by the extension of the redness over the soft palate and by the tumefaction of the tonsils, but the skin also begins to present lesions. According to my observations, the eruption, as a rule, makes its appearance on the first day of the disease, in a decided minority of the cases on the second day, and in rarer instances at a later period. Generally it is recognized at about the same time on the neck, chest, and lateral parts of the face and forehead, but especially on the parts first mentioned, in the shape of numerous red points situated close to each other, and spreading rapidly over the rest of the body. On the face and forehead the eruption may be confounded with the flush produced by fever. When the cheeks are much reddened by the eruption, the very pale color of the region of the mouth presents a striking contrast. It is only exceptionally that the face is entirely free from the eruption. The skin has often a tense or turgescent appearance, and sometimes the eruption is preceded by a slightly erythematous coloration of the skin over a greater or smaller surface. As a rule, the extension of the eruption over the whole body occupies scarcely half a day. The rapidity of its progress is greater than in other acute eruptive diseases.

The time required for the full development of the eruption is very variable; in mild cases the eruption occasionally reaches its height on the evening of the first day; in moderate on the second; and in severe cases on the third or fourth day; in rare instances even later. When the maximum occurs early the eruption is punctiform, each minute point being surrounded by pale skin; when the maximum occurs later the eruption becomes more and more confluent, until finally, when it reaches its full development, the neck and body, as well as the greater part of the face and extremities, present an intense uniform scarlet red color, with here and there isolated points of a darker appearance. At the same time the skin is very hot, generally dry, sensitive to the touch, tense, swollen, and the subcutaneous cellular tissue in various places, especially the cheeks and eyelids, œdematous. In cases of moderate severity these symptoms are not well marked, and in mild cases the skin may be normal, with the exception of redness and a moderate increase of temperature. Sudamina often make their appearance at this time; in some cases the vesicles are numerous, in others scanty, while in the milder forms of the disease they are generally absent. In severe cases, where the eruption is copious, small extravasations may be seen in some places, especially about the mouths of the hair follicles.

As the eruption progresses to its full development, the other symptoms increase or remain unchanged; the tip of the tongue becomes very red, and the coating thicker; the latter often now peels off in places, but does not usually disappear completely before the fourth day, after which time the tongue presents for several days a characteristic appearance. It is red, smooth, dotted with warty swollen papillæ, and is often somewhat dry, as are also the lips; the affection of the throat increases, and the tonsils, which are enlarged, are covered with a yellowish-white layer of thick pus, or even with a small amount of true diphtheritic membrane. The appetite is now quite gone, the vomiting has by this time generally ceased, the bowels are constipated, but occasionally there is slight diarrhœa with colicky pains. The fever increases, if it have not attained its height at the outset of the disease, and now reaches the neighborhood of 105.8°, keeping at this point with slight variations in severe cases, and with greater variations in the mild ones. In some instances, on the morning of the second day of the disease, before the full development of the eruption, the temperature shows a considerable remission, or even a complete intermission, and this although the case is otherwise of the usual severity. The pulse is usually very frequent, and may become so rapid that it cannot be counted; now and then, however, without any obvious cause the frequency is only moderate. Occasionally there are chest symptoms, such as cough, pain, etc.; these are generally indications of the beginning of some complication. The moderate dyspnœa, which is present, is often due to the fever. In the same way are to be explained certain cerebral symptoms, such as more or less severe headache, restlessness and mild delirium at night, sometimes wandering of the mind by day, but more commonly somnolence. The urine at this period is scanty and concentrated, and contains a very small quantity of blood, or is albuminous, especially if the fever be high. This shows that even at the outset of the disease the kidneys are unmistakably involved, although only in the form of a catarrh of the renal tubules.

The eruption continues at its maximum only a short time—from a few hours to half a day, or a whole day—and then begins to fade, first upon the upper part of the body, and afterwards gradually, but still with some rapidity, upon the lower portions. The last traces of it are to be seen in the broad and slightly infiltrated roseolæ on the back of the hands and feet, and in the sudamina, if there have been any. In normal cases the period of decline occupies from two to three, or even four days, making the whole duration of the eruption from three to seven days. During this period desquamation commences. The earliest, but not the usual form of it, is the furfuraceous; the more common form is the lamellar, which occurs somewhat later and follows as a rule the other variety. In normal cases, while the eruption is fading the general symptoms subside; the fever moderates, but not so rapidly as to constitute a crisis. The defervescence takes place generally in the form of lysis, with gradually lengthening remissions, and afterwards shortening exacerbations, so that it takes several days before the normal temperature is reached. The frequency of the pulse falls to the normal, following the course of the temperature, the skin becomes moister, loses its turgescence, and at this time sudamina are very apt to form. The swallowing becomes gradually easier and less painful, the redness and swelling of the tonsils subside, and the membranes, if there are any, become detached; the tongue becomes more moist, and by the end of this period the superficial layer of epithelium is regenerated. Rhagades now heal, the appetite continues to improve, digestion becomes normal, the urine more abundant and clearer, the extraneous substances from the renal tubules and the albumen disappear. With the fall of the temperature the cerebral symptoms gradually subside, the sleep becomes quiet and refreshing, and the patient consequently regains his strength. A week after the height of the eruption, or a week and a half after the beginning of the disease, convalescence is generally complete, if the case have been of moderate severity and uncomplicated; but caution is necessary for a long time after this.

The greatest care, however, will not always prevent the occurrence of farther troubles, which may arise both during the attack and during convalescence, and may affect different parts of the body; I refer to the *other localizations of the scarlatinal poison*, the affections of the lymphatic glands and the joints, and the disease of the kidneys. Even when the scarlatina has run a perfectly normal course in respect to the eruption, angina, and fever, our hopes of a favorable convalescence may be dashed at any time and under any treatment by the occurrence of one of the above-mentioned complications, or by several of them appearing together or following each other.

In the majority of cases of scarlet fever most of the *lymphatic glands*, which are accessible to examination, especially the *cervical*, *i.e.*, the submaxillary and jugular glands, are more or less swollen and painful. It is altogether probable that this swelling is due to the special influence of the throat affection, yet it is to be noticed that there does not appear to be a definite proportion between the two lesions; there may be, for instance, severe angina with slight glandular affection, or, on the other hand, considerable lymphadenitis, with a trifling amount of angina. When the course of the eruption and angina is normal, the moderate enlargement of the lymphatic glands disappears imperceptibly; in other cases, just as these symptoms are beginning to subside, or when they have already vanished, and desquamation is in full progress, about the middle or the end of the second week there occurs a more or less rapid proliferation of the cervical glands, sometimes equally on both sides, sometimes more on one side than on the other, and at other times only on one side, with, perhaps after several weeks, an extension to the other. The fever, which may have already disappeared, now generally returns in greater or less force, or if it had only begün to subside it becomes more intense; the restoration of the appetite and health is interrupted, and possibly further derangements or actual complications may be produced.

The *affection of the joints* in scarlet fever usually takes place during the decline of the eruption, when desquamation commences; and this is the case whether the course of the disease has been mild or severe; it generally lasts only a few days. With or without increase of the fever, pains occur in one or more joints, sometimes only in the small joints of the hands and feet, at others also in the larger joints. The affection usually runs much the same course as ordinary rheumatism of the joints, except that ordinarily it does not last so long; the pain increases rapidly, but disappears after a few hours, to reappear in other joints, so that within a few days all the joints of the body may be affected once. Although the anatomical changes are so slight as to be scarcely noticeable, the pains are very severe, at least children often complain bitterly. Scarlatinal rheumatism is not always of short duration; sometimes it fastens itself upon one or more joints, particularly the larger ones, and gives rise to intense synovitis, with more or less considerable fluid accumulation, in fact even to more serious chronic lesions. In such cases the fever generally runs high without pursuing a definite course, but rather varying as it does in ordinary polyarthritis. The other symptoms are similar to those of this latter affection, especially the perspiration, which in scarlatina leads to the formation of numerous sudamina. The same resemblance holds in regard to the complications.

Nephritis occurs in scarlatina at various periods, with different symptoms, and probably depends upon a variety of causes. In some cases its existence can be ascertained only by the examination of the urine, since the normal course of the scarlatina terminates in recovery; the most certain evidence is, of course, to be obtained by direct examination of the kidneys, if death occur suddenly during the normal course of the disease through an unexpected accident. Up to the present time there has been no record of such a case, and in the absence of any or at least of characteristic signs of disease as revealed by the urine (and they certainly are absent in very many normal cases), we cannot tell whether there may not be more or less marked pathological changes in the kidneys notwithstanding the lack of symptoms. The urine in hydrops scarlatinosus (always a result of nephritis) is scanty, and so loaded with urates as to make the microscopical examination very difficult. In rare instances the urine has been found to be free from albumen and casts; we are consequently not to infer that the kidneys are healthy because the secretion is abundant and apparently healthy, with no uric acid deposit, as is the case at the beginning of the eruption, and when the course of the scarlatina is normal.

It is different, however, in those irregular cases which frequently prove fatal at an early period of the disease; here the autopsy often reveals disease of the kidneys.

Steiner found as a result, he says, of numerous careful observations, that in scarlatinal patients, who died early, there was always more or less hyperæmia of the kidneys, and that in other cases, besides the hyperæmia of the kidneys, there was catarrh of the renal tubules to such an extent as to present the signs of diffuse nephritis in its various stages; in one form or the other the kidneys were always diseased. In the children who died of scarlatina on the second or third day, and whose kidneys were found to be hyperæmic and enlarged, with cloudiness of the epithelium of the renal tubules, the urine, so Steiner states, gave no evidence during life of kidney disease. In another place he expresses the opinion that in the slight catarrhal affection of the renal tubules, which remains as a mere catarrh without progressing to its further development, the urine is either normal or contains merely traces of albumen and desquamated epithelium; while the diffuse catarrhal form of the disease manifests itself at an early period by larger quantities of albumen and epithelium. A catarrh of the renal tubules which reaches this extent generally terminates sooner or later (as a rule between the thirteenth and twenty-first days of the disease) in "Morbus Brightii" (croupous or parenchymatous nephritis); the urine is not only diminished in quantity, but contains albumen, desquamated epithelium, fibrine from the blood, and disintegrated casts, the quantity of these ingredients varying with the extent of the parenchyma involved. The development of the nephritis out of the catarrh of the renal tubules, resulting partly from mechanical obstruction, by means of the abundant desquamation of epithelium and the consequent stasis of the blood in the kidneys, coincides generally with the desquamation of the epidermis; and this fact has led to the erroneous opinion that the affection of the kidneys is a complication of the stage of desquamation, whereas the careful early examination of the urine shows that this is not the case. Croupous nephritis, in rare instances, may arise suddenly without being preceded or ushered in by a catarrh, and is then generally fatal. In regard to this question of the development of parenchymatous nephritis, I wish to again call attention to the fact, that in normal cases up to from the thirteenth to the twenty-first day the proof of the existence of a renal catarrh is generally obtained only accidentally, and that in the absence of morbid signs on the part of the urine, it is probable that there is no catarrh, but merely a partial derangement so trifling as not to present any symptoms. Even in severe cases, as long as the affection runs a normal course, the symptoms which, during the eruption and height of the fever, indicate a renal catarrh, usually disappear entirely when the severe symptoms of the disease subside, and they may disappear also even when renal symptoms due to parenchymatous nephritis arise between the thirteenth and twenty-first days. This explanation of the origin and genesis of parenchymatous nephritis in scarlet fever, however interesting it may be, is still not entirely satisfactory; and from the mere fact of the existence of insignificant changes in the urine, such as the small amount of albumen and desquamated epithelium, which are often seen at the outset of severe cases, whether normal or abnormal, we are not justified in concluding that they are due to a specificness of the disease, and not rather to the fever. It is a matter of common observation that in other infectious diseases, as well as in those which are non-specific, such changes are undoubtedly produced by the febrile condition. It is probably safe, therefore, to infer that many of the lesions of the kidney occurring in rapidly fatal abnormal cases with symptoms of intense fever, are the result of the fever, and not of the scarlatina; but this is a theory more difficult of proof than the one first mentioned.

The examination of the urine gives, according to my experience, the following result: At the beginning of the disease, especially during the development and height of the eruption, the character of the urine varies considerably. It may not only be free from albumen, but very often exhibits no indication of catarrh of the renal tubules. Not unfrequently, however, the urine, although in other respects clear and free from albumen, soon begins to show a mucous cloud, which is composed of more or less abundant cloudy and degenerated epithelium, and, sooner or later, cylindroidal, in rare instances epithelial, and still more rarely hyaline casts. The cylindroidal casts are very long, and look like cylinders; sometimes like smooth, narrow bands; sometimes quite broad bands with longitudinal striæ and fringed extremities; at other times the casts are narrower, cracked transversely, split up, in fact present the appearance of casts which have collapsed; sometimes they look as if composed of minute fibres. At this period albuminuria occurs only exceptionally, and then only to a slight amount and for a short time. When it does occur the urine generally contains red and white blood-corpuscles in addition to the epithelium and cylindroidal, epithelial, and hyaline casts. Catarrh of the renal tubules is a frequent, but by no means constant accompaniment of the eruption, and when it does occur its symptoms are generally of the mildest character, and rarely of a serious nature at this period of the disease. In no case does the kidney affection, when at all severe, develop at once into its full intensity, as is sometimes the case with the eruption; on the contrary, it is scarcely noticeable on the first day, and gains headway gradually, first during the progress of the eruption. When the eruption fades and the fever subsides, the renal catarrh generally declines also, so that in the second week it may have entirely disappeared; but it may continue in diminished intensity up to the time when the more serious lesions of the kidney are apt to occur.

It is thus apparent that the relation of the catarrh of the renal tubules to scarlet fever is quite different from that of the bronchial catarrh to measles. In the latter case the two affections correspond to each other, but between measles and scarlatina there seems to be no correspondence, either in respect to the regularity of the catarrh or in respect to the occurrence of this symptom in connection with the initial fever, the eruption, or the decline of the eruption.

The renal catarrh, which occurs at the outset of scarlet fever, is certainly not completely identical with those special forms of like character which occur more or less uniformly in other infectious diseases, such as cholera, typhoid fever, petechial fever, and relapsing fever. Each of these diseases has its peculiarities, especially in the situation of the local affection, which is sometimes in the parenchyma, at other times in the tubules of the kidney.

On the other hand, the condition of the urine in various other acute febrile diseases is in the highest degree similar to what is found in scarlet fever. In croupous pneumonia, measles, meningitis, intestinal catarrh, in short, in a great variety of affections, I have often found the same cloudy epithelium and the same epithelial, hyaline, and cylindroidal casts as are found at the beginning of scarlet fever. In these diseases renal catarrh does not occur so frequently, and this fact seems to justify the conclusion that the catarrh of scarlet fever is due to a specific irritation, as well as to the febrile condition. In a given case sometimes one cause seems to be more active, sometimes the other.

In those cases of scarlet fever which present symptoms of parenchymatous nephritis at the usual time, *i.e.*, after the conclusion of the eruptive stage, towards the end of the second week at the earliest, generally during the third or at the beginning of the fourth week, the changes in the urine are much more striking than at the beginning of the disease. During the first few days, in the most favorable cases, the abnormal constituents are trifling in amount, or perhaps entirely absent; then cylindrical casts gradually make their appearance, and afterwards an abundance of more or less cloudy and degenerated, or perhaps merely swollen or quite normal epithelium, with granular detritus, and red and white blood-corpuscles. These admixtures make the secretion turbid, and produce sediments of a grayish white or dark color. At this time hemorrhage from the parenchyma of the kidneys is a common occurrence, and the urine consequently becomes more or less reddish-brown and very turbid, and usually contains an abundance of epithelium and casts. The secretion now becomes much diminished, sometimes almost suppressed. Baginksky states that sometimes, though rarely, the stage of diminished secretion is preceded by a temporary diuresis. Usually the urine is very concentrated and deposits urates in such quantity as to interfere with a satisfactory microscopical examination. The chemical examination, which up to the middle or end of the second week, has perhaps entirely failed to disclose any abnormality, now shows the presence of albumen in quantities which increase from day to day. In my experience the quantity generally remains small if it makes its appearance first at the end of the third or early in the fourth week, but is more abundant if the renal affection occur earlier. The amount of albumen does not always correspond to the quantity of blood and casts, yet this is the general rule. Exceptions, however, do sometimes occur; occasionally, though rarely, numerous casts may be seen, and yet for days together no trace of albumen; on the other hand, a considerable albuminuria may be present while the urine is perfectly clear and free from sediments, and even the filtrate shows nothing abnormal. The examination of the urine, when the renal lesion is trifling, is thus evidently untrustworthy, and it is consequently impossible to date the beginning of the parenchymatous nephritis.

Very often individual peculiarities may be noticed in respect to the quantity and quality of the abnormal ingredients of the urine; for instance, in one patient the urine up to the beginning of the third week is clear, in others it is for a long time uniformly turbid, and contains cylindroidal casts. In the first case, when the parenchymatous nephritis sets in, numerous epithelial and hyaline casts may be seen; in the second case, besides these abnormalities there are also the transitional form of the cylindroidal and a few epithelial casts; but in the third case the cylindroidal casts disappear at once and permanently. Sometimes the casts are studded with detritus; sometimes they also contain cells of every variety imbedded in them, and at other times they are entirely free from formed elements, etc. The renal symptoms are as varied in character as are those of the eruption and other organic affections.

Eisenschitz and Steiner dissent from my account of the origin of nephritis scarlatinosa in important particulars. The former believes that scarlet fever does not occur without catarrh of the renal tubules, any more than measles without catarrh of the respiratory passages. He claims to have discovered the renal affection when the eruption has existed less than twelve hours, and insists that cases without catarrh, and especially albuminuria, are very rare. The renal catarrh may continue as such during the whole course of the scarlet fever, varying only in intensity, or it may become the starting-point for other more serious lesions of the kidneys, such as chronic Bright's disease, in which the urine is clear, abundant, and contains but little albumen, and acute Bright's disease, in which the urine is reddish-brown, turbid, very albuminous, and scanty. The latter variety, he says, may arise either directly from the catarrh or from the "chronic" affection. The difference between us is this: it is only rarely that I am able to find any symptoms of catarrh at the outset of the disease, and when they do occur, I see them generally diminish with the decline of the fever in the second week, or disappear entirely; and finally, without any connection with such symptoms at the beginning of the disease, I see a new affection of the kidneys breaking out with more or less suddenness, while Eisenschitz always finds a catarrh from the beginning to the termination of the disease. It is certainly singular that he gives us no detailed description of the symptoms which occur during the transition from catarrh to nephritis.

It is possible that these differences may depend upon local and epidemic peculiarities.

Although the alterations in the urine are the most important indications of scarlatinal nephritis, there are other symptoms which are quite significant. The patient complains of pain in the region of the kidneys, voluntarily, if he be old enough, but only on pressure if a young child; the appetite diminishes; the abdomen becomes tense and swollen, perhaps even painful, or there is at least a feeling of fulness, and not unfrequently also vomiting, constipation, nausea, headache, sleeplessness, restlessness, and peevishness. In the second week, when the fever has moderated considerably, or has quite disappeared, it may return in variable intensity with the occurrence of the symptoms of renal disease. In mild cases this relapse does not occur, or is limited to slight exacerbations in the evening; and even in severe cases with high temperature, the fever is short or quite ephemeral. At this time a moderate amount of œdema also makes its appearance, generally only on the face, particularly in the eyelids, producing more or less bloating, also about the ankles and on the buttocks, sometimes only on one side.

These initial symptoms, as a rule, continue in the same intensity for a variable length of time; sometimes, however, they diminish somewhat in severity, and in rare instances, even when the case is otherwise normal, they become aggravated. In the latter case the urine becomes scanty, very albuminous or bloody; there is pain in the region of the kidneys and on micturition; the œdema increases to a moderate extent, the slight fever, cerebral disturbance, and vomiting continue; a more or less severe diarrhœa sets in, and pulmonary symptoms make their appearance, such as catarrh, especially over the lower lobes, distressing cough with a moderate amount of dyspnœa and laryngeal affection. Although there is some fever the pulse very commonly at this time becomes remarkably slow, from fifty to sixty beats per minute in children, and under fifty in adults, soft, and often irregular; the first sound of the heart is diffuse or replaced by a slight murmur. After these severe symptoms have lasted for several days improvement is indicated by the cessation of the fever, return of the appetite, and especially by the increase in the amount of urine, which now becomes clearer and of lighter specific gravity; it loses its dark color, no longer contains blood or precipitates sediments, and the albumen steadily diminishes in quantity from day to day. The desquamation of epithelium in the renal tubules also gradually ceases, the casts lose the imbedded cells, detritus masses, etc., and become less numerous, and at the same time the amount of grayish-white sediment in the urine decreases. Sometimes the œdema now disappears entirely, the pulse regains its normal frequency, the heart's action becomes more vigorous, and the patient, enfeebled by long disease, recovers his strength. The desquamation of the skin, which had remained up to this time more or less incomplete, often now goes on with renewed activity.

It is difficult to ascertain with accuracy the duration of the nephritis in normal cases, partly because it is not easy to distinguish between normal and abnormal cases, but principally because in tolerably severe cases the morbid microscopical elements in the urine continue to appear a long time after the albuminuria has ceased. Weeks after the disappearance of the last trace of albumen we find single or even moderately numerous hyaline casts, with or without cloudy epithelium, detritus, and red and white blood-corpuscles; showing that, at least, in some parts of the kidneys the inflammatory process still continues. As a rule, it is safe to say that the normal duration of the albuminuria is about two or three weeks, and that casts are discharged for about one month. Besides these severe cases with prolonged albuminuria, mild cases of nephritis very often occur, in which the morbid symptoms last only a few days, and also abortive cases, in which the renal affection disappears still more rapidly.

Irregular Course of Scarlet Fever.

Scarlet fever in its irregular forms may terminate favorably or unfavorably, and its symptoms in such cases may present every variety from the mildest to the most severe character. Following the excellent division of Wunderlich I divide the irregular forms of scarlet fever into those with rudimentary and those with fully developed localizations.

Of the *rudimentary*, and therefore *mild* and *favorable* forms, the most frequent is simple scarlatinal angina. A slight or moderately severe fever of short duration, accompanied by some pain in the neck and enlargement of the cervical glands, is not an uncommon occurrence in persons who have but little predisposition to the disease, especially if they be of mature years, and also in those who have already had scarlet fever in childhood. These symptoms are the more suspicious if scarlet fever have occurred in the family, or if the patient have probably been exposed to the poison. If the throat be examined, the characteristic redness is seen in a mild form, with or without a moderate enlargement of the tonsils; perhaps also the tongue presents the appearance of the scarlet-fever tongue. There are also malaise, anorexia, headache, and other symptoms of slight importance. Such attacks generally disappear in a few days, but they should receive the same attention which is paid to the unmistakable disease; every throat affection during a scarlet-fever epidemic is suspicious!

In other cases the angina is absent, or so trifling that it does not account for the existing fever. When present it lasts only a few days, with the symptoms of a slight febricula. That it depends upon infection with the scarlatinal poison, is evident from the above-mentioned circumstances; but at other times the diagnosis is confirmed by a simultaneous enlargement of the cervical lymphatic glands, by a suspicious redness of the skin, a slight affection of the joints, albuminuria, and other renal symptoms. The redness of the skin is generally too uncharacteristic to warrant a positive diagnosis; on the face it may be mistaken for an unusually marked flush from fever, in other parts for an accidental result of pressure, etc; in many cases it is merely a scattered roseolar eruption, which looks like measles and lasts only a short time, sometimes merely a few hours.

All these varieties of the disease are frequently of such short duration, and apparently of such trifling importance, that the patient pays but little attention to them, and often entirely overlooks them. But in other cases the fever and local symptoms are more serious, and continue so long as to constitute a sort of transition to the usual forms of the disease, the eruption only being absent. In the absence of satisfactory ætiological evidence, the scarlatinal nature of the attack is proved by the subsequent occurrence of the characteristic desquamation, even when there has been no previous trace of an eruption, and by the appearance of a moderate amount of dropsy and albuminuria. These cases may be designated as Angina scarlatinosa, Nephritis scarlatinosa, Febris scarlatinosa sine exanthemate sive sine scarlatina.

There is also an unusually mild course of the disease, in which the eruption is very scanty, poorly developed, and confined to certain parts of the body. In these cases the angina may be absent, or so trifling in amount as to be readily overlooked. After a slight fever lasting for several days, during which there are some insignificant local affections, these insignificant symptoms disappear entirely, or are followed by a rudimentary rheumatism or a scarcely noticeable anasarca.

Finally there are other cases, in which, besides the fever, which in all probability depends upon scarlatinal infection, other *isolated* derangements arise, which are not commonly present in scarlet fever, and are rather of the nature of complications; such as gastro-intestinal catarrh, parotitis, slight cardiac affections, etc. The diagnosis of this variety must be based upon ætiological considerations.

For the same reasons we may here include also those cases in which more or less rudimentary scarlatinal symptoms occur in the course of other diseases; according to Wunderlich, local hyperæmias of the skin, especially of the palms of the hands and the chest, with subsequent desquamation; angina in patients who are rarely affected with it; a peculiar redness of the tongue under the same circumstances, etc.; and also other symptoms which are more fully developed but still rudimentary.

Rudimentary affections sometimes occur which are of a more serious nature. They are characterized by essentially the same symptoms as the favorable varieties of the disease; by a rudimentary eruption without angina, angina without eruption, but also by severe cerebral disturbance with very high fever, and often collapse. Sometimes these malignant cases begin in a mild or normal manner, and then, after the disease has progressed for a time, without the slightest occasion for alarm, symptoms suddenly arise of the most dangerous character, and death speedily ensues. Usually, however, severe symptoms are present from the start; intense headache, very great restlessness, marked drowsiness or actual coma, profound malaise, severe convulsions, which do not appear to depend upon the intensity of the fever nor upon local disease; even severe tetanus, trismus, and other local tonic and clonic convulsive movements, persistent vomiting and diarrhœa, severe dyspnœa, which seems unwarranted by the condition of the respiratory organs, troublesome rheumatoid pains over the whole body, extreme rapidity of the pulse, which is small and scarcely perceptible to the touch, and symptoms of collapse. Before the disease has fairly developed itself and exhibited its scarlatinal origin by characteristic local symptoms, death often occurs on the very first day, either in an intercurrent fit of convulsions of unusual severity, or, in the absence of these, with the signs of intense fever or profound collapse. Gläser describes such a case which proved fatal within eight hours, Bohn and Hambursin one within ten hours, Trousseau in eleven hours, Stiebel two cases in twelve hours, Epting one in fifteen hours. The autopsy in these cases usually reveals lesions which are quite insignificant; the blood within the vessels is thin and of a bright or dark red color, capillary hemorrhages are seen scattered in more or less numerous places over the whole body, the brain is hyperæmic, and the sinuses distended with blood; there are also commencing meningitis, a flabby heart, enlarged spleen, and some congestion of the kidneys. The alarming symptoms which occur during life are to be explained by the severity of the infection, and should be regarded as indications of intense "blood-poisoning." In such cases the diagnosis is often doubtful, even after the autopsy, and we are obliged to rely upon the evidence of previous exposure. In some instances scarlet fever proves so rapidly fatal, with convulsions or other severe cerebral symptoms, only because it happens to occur at a time when there is already existing brain disease, such as tuberculosis or chronic hydrocephalus, predisposing to dangerous symptoms of this character.

Finally there are other cases, terminating after a longer or shorter time in recovery, which present the following history: The eruption and other characteristic local lesions are absent, but the attack is a severe one on account of certain brain, lung, stomach, or intestinal symptoms of a suspicious character, or also on account of the duration and height of the fever, or, in the absence of any clearly marked local symptoms, merely on account of the intense fever. After a longer or shorter time recovery takes place, and the doubt is cleared up sometimes by the fact that the symptoms occur in connection with unmistakable cases of scarlet fever, and sometimes by the subsequent occurrence of affections which are characteristic, such as desquamation, nephritis, and dropsy. They vary in severity, and seem to constitute a variety intermediate between the abortive forms and the malignant cases with rudimentary local symptoms.

The irregular forms of scarlet fever with *fully developed local lesions* may be divided into two classes; in the first, the course of the disease is at first perfectly normal, and the irregular symptoms occur at a later period; in the second, the latter arise at the outset of the attack. In both classes the irregularity may affect either the local process or the febrile symptoms; if the former, the irregularity is noticed only in some of the affected organs, or in all of them at the same time, or in one after the other; if the latter, the fever is more intense than usual, or more protracted, or both together. In the abnormal course true complications are not necessarily present, but they frequently do occur, and then change the character of the disease still farther. In some instances they give rise to no noticeable symptoms, and are first recognized, after the disease has run its course, by the sequelæ or by the fatal termination; but even the autopsy often fails to reveal any complication. Death is quite a frequent result, occurring at almost any period of the disease, but of course always later than in the variety with rudimentary local lesions. The most malignant epidemics are those in which the abnormal cases occur the most frequently.

When the course of the disease *begins normally*, and *becomes irregular subsequently*, the normal period lasts a variable time, generally at least several days.

When the fever is irregular it fails to defervesce after the normal progress and disappearance of the eruption and angina, but continues for weeks, sometimes with the same intensity and with a typhoid character like that of a variety of scarlet fever to be presently described, sometimes with increasing intensity, especially if it is to prove fatal, and at other times it declines gradually, as in protracted defervescence. In such cases the pulse is often very rapid, the heart's action violent, and the first sound of the heart diffuse or even replaced by a distinct murmur.

This murmur is not necessarily of serious importance; it may be merely a blood murmur, but sometimes actual regurgitation occurs for a time, owing to paresis of the papillary muscles, without valvular lesion (accentuation of the second pulmonary sound). Partly in this way, and partly as a result of disease of the whole cardiac muscle, the heart may become dilated and its area of dulness increased. True endocarditis at this period of the disease, especially of the mitral and aortic valves, and myocarditis belong to the complications of scarlet fever.

In consequence of the exhaustion resulting from intense fever there not unfrequently occurs with a normal temperature a severe form of *nervous delirium*, attended by motor disturbance, and often presenting the character of melancholia, or delusions of persecution; in rarer instances, of extraordinary gayety and talkativeness (Steinthal). These symptoms generally disappear in the course of a few days, or earlier, after a good sleep procured artificially or occurring naturally.

Independently of existing fever, irregularities may occur in the course of a hitherto normal scarlet fever, in the shape of *intercurrent nervous attacks*, which unexpectedly arise from some external cause or spontaneously, as, *e.g.*, convulsions of all kinds, tetanus, spasm of the glottis, hysterical and maniacal symptoms, delirium, jactitation, general excitability, dyspnœa without apparent cause, and extreme rapidity of the pulse.

Sometimes while the disease is progressing normally, the patient suddenly sinks into collapse without known cause, or apparently after exposure to cold or after a purge; the skin becomes pale and cold, the eruption disappears, the pulse becomes thready and uncountable, and death ensues.

Finally, after a normal course of the first local symptoms, and even of the fever, there may occur at any time in severe cases a *hemorrhagic diathesis* terminating either in recovery or death.

Irregularities may arise, moreover, in connection with the *local lesion*. Under this head the following varieties are worthy of attention:

Irregularities in the *eruption*. This may become hemorrhagic when its development is intense; or, upon the surface of the skin occupied by it other eruptions may arise, vesicles, pustules, etc.; or the mode of desquamation may be irregular, too abundant, repeated, etc.

Irregularities of the *angina*. Instead of declining with the eruption, it may increase considerably; the tonsils may become much swollen, and may suppurate or become gangrenous; diphtheria may set in at any time, either upon the much inflamed parts of the throat, or after the original angina has partially or wholly disappeared.

Irregularities in the course of the disease of the *cervical lymphatic glands*. In the ordinary course of this affection the usually moderate swelling subsides, but sometimes the inflammation extends to infiltration of the cellular tissue of the surrounding parts, and abscess results. Abscesses may occur upon one or both sides of the neck, at the same time or in succession. They seldom heal rapidly, but generally slowly, as glandular suppurations ordinarily do. Death in rare instances results from the exhaustion of suppuration. Sometimes the glandular affection terminates in chronic induration and its results.

Rheumatism of the joints sometimes runs an abnormal course terminating in considerable synovitis.

The most important irregularities are those which are caused by *renal inflammation*, which may occur in any case, the mildest as well as the most severe, and without any connection with the mode of desquamation or the condition or functional activity of the skin. It appears at the same time as under normal conditions, sometimes during convalescence, sometimes during the continuance of the fever or just before its complete disappearance when it has been prolonged by one of the above-mentioned normally developed localizations. The initial symptoms of the nephritis may be the same as those described in the normal course of scarlatina, but their irregular character may sometimes be disclosed by the greater severity of some or all of them, by the fever, the scantiness and character of the urine, especially the dark color produced by various coloring matters, *e.g.*, bile pigment and hæmatine. At this time there gradually occur marked dropsical symptoms, which may sometimes be predicted by the increase of weight which is apparent even to the attendants upon the patient, and is not to be accounted for by increased nutrition. Except in cases where there is a certain amount of œdema from profound anæmia after a very severe course of the disease, there will always be found to be kidney disease; in fact, there is no scarlatinal dropsy without nephritis. Moreover, the dropsy need not necessarily be preceded by albuminuria; the latter may be entirely absent or exist in a very different degree of intensity from the intensity of the dropsy. It is evident that the dropsy is connected with some affection of the vaso-motor nerves, and not dependent alone upon changes in the kidneys and loss of albumen. With the diminution in the quantity of urine, the dropsy soon spreads over the entire surface of the skin, or a great portion of it, especially over the lower extremities, the genitals, and the face, where it often reaches the highest grade. Thus the anasarca often develops so rapidly that a miserable and badly nourished child may in a single day present the appearance of an excessively fat one. During its continuance the œdema is subject to frequent changes. At the same time with or after the anasarca, further effusions generally make their appearance, such as organic œdemas and infiltration of the submucous cellular tissue, but especially effusion into the serous sacs and into the ventricles of the brain. The most common of these lesions is ascites, which is accompanied by excessive and painful distention of the abdomen; it often reaches a considerable degree, and then causes important functional disturbance, for instance by compression of the intestines, and interference with the respiration by pressure upon the diaphragm. In fatal cases the collection of serum is generally of a pale yellowish color, clear, and contains much albumen; the peritoneum is not injected. Hydrothorax is less common, but sometimes makes its appearance at a very early period, with considerable anarsarca, and sometimes also at a late period, as a rapidly fatal complication, accompanying excessive dropsy with ascites. Both pleural cavities are not always attacked, but often only one. Œdema of the lungs is often present at the same time; it sometimes comes on at the beginning of the nephritis, unnoticed and gradually; sometimes suddenly at the height of the disease, and then generally as a sign of impending death, and as its immediate cause. Hydropericardium seldom occurs isolated, but is rather a local manifestation of the general dropsy; when it reaches a high degree it causes marked dyspnœa and quickening of the pulse, but no other special symptoms. The œdema extends also to the uninflamed mucous membranes; for instance, to the soft palate, the uvula, the neighborhood of the ligamenta aryepiglottica (œdema glottidis); in the latter case, speedy death by suffocation is almost certain. In the cranial cavity the serous effusion manifests itself sometimes by true œdema of the brain, sometimes by hydrocephalus externus, more rarely and in a low grade by hydrocephalus internus, or hydrops ventriculorum. These several lesions are generally only partial symptoms of a general anasarca; sometimes, however, they occur as isolated symptoms in the form of hydrothorax or slight ascites, but perhaps more often as œdema of the brain. Frequently the serous effusions all take place simultaneously; generally they follow, or, to a certain extent, complicate each other; in reality, however, they are only an expression of the advance and severe course of the disease.

The frequency of dropsy and albuminuria in epidemics of scarlet fever is very variable; some writers affirm that nearly all the patients are dropsical, others that such cases are very exceptional. According to Steiner, the frequency of nephritis parenchymatosa in the several epidemics varies between five and seventy per cent.; according to others it may appear less often, but also even more frequently. Dropsy does not often occur before the middle of the second, and sometimes not until the end of the fourth week; still more rarely at even a later period (according to Russegger, once in the ninth week), but generally in the third week. It is said that the dropsy of scarlatina may make its appearance even after several months have elapsed.

During the continuation of the dropsy the urine is generally rather scanty, of high specific gravity (1.025–1.040), more or less turbid, rich in albumen and casts, sometimes also quite bloody; less often cases occur where the urine remains normal, or at least clear and free of albumen. In regard to such cases it has been remarked not only that albuminuria is absent in certain patients at a particular time of day, or during several days, but also that this may be the case in a large number of patients in an epidemic, and there have even been epidemics of scarlet fever in which all the dropsical patients were free from albuminuria. But in most epidemics dropsy, without albuminuria, is a rare occurrence.

Among the other symptoms of dropsical patients the following are worthy of notice: anorexia, frequent vomiting and diarrhœa, and other gastro-intestinal derangements. Not infrequently the irritability of the mucous membrane of the digestive organs outlasts the dropsy. The patients complain of cough, pain in the side, and dyspnœa, which are due to bronchial catarrh or more serious chest lesions; there is headache, sometimes drowsiness, or insomnia and excitement; the liver and spleen become swollen; in addition to the early dropsy there are generally to be found the usual affections of the lymphatic glands and auditory apparatus. The skin, in consequence of the anæmia and infiltration with serum, presents a peculiar waxy paleness and a somewhat transparent appearance; at the time of fever it is dry and brittle, later it is often moist and covered with miliaria. The heat of the body varies; sometimes it is entirely normal, even when the course of the nephritis is abnormal; more often it is elevated during the whole evening, and returns to the normal by morning. Sometimes, especially at the outset, there is a distinct elevation; in other cases there are marked intercurrent exacerbations, of short duration, in the midst of a normal or slightly elevated course of the temperature, and in rare instances the temperature remains high for a considerable time. A notable peculiarity of scarlatinal nephritis, namely, the retardation and moderate irregularity of the pulse, is often observed in the dropsical cases, even when there is considerable fever, provided the pulse be not quickened by the approach of death, or by some accidental cause. The scarlatinal dropsy is often fatal; when it runs the course above described the fatal result is generally due to anæmia, in consequence of gastro-intestinal disturbances, or to severe affections of the respiratory organs.

In the most severe cases—especially in those where the secretion of urine is very scanty, or entirely or almost entirely suppressed for several days, or even exceptionally for a week or ten days—uræmic symptoms often occur, sometimes with the presence of considerable dropsy, sometimes with only a slight amount, or none at all. When there is much anasarca the uræmic symptoms may display themselves in the form of stupor, with headache lasting for some time — fourteen days, according to Zehnder; but at other times convulsions break out, preceded by such serious symptoms as very intense headache, vomiting, amblyopia, and even complete blindness, tinnitus aurium, perhaps even delirium and coma, and this after transient improvement had taken place and given rise to false hopes. The convulsions sometimes attack only certain groups of muscles (trismus); sometimes in an exceedingly varied way the trunk and extremities; sometimes only one-half the body; they may also be perfectly epileptiform, or tonic and clonic spasms may alternate at one and the same place during the attack; sometimes there may be only a single attack, which may last from five minutes to several hours; but not unfrequently several attacks follow each other in the course of a few days; exceptionally, numerous slight attacks occur on a single day after intervals of varying, generally short, duration. During severe uræmic attacks death suddenly supervenes from œdema of the lungs, stoppage of respiration, exhaustion from too long-continued and too oft-repeated severe convulsions, cerebral hemorrhages, etc.; or after the convulsions have ceased death may occur gradually in other ways by collapse from paralysis of the heart, by œdema of the brain, or by hydrocephalus. But if at this period the disease incline to recovery, the convulsions cease, and the profound stupor and severe brain symptoms which existed during their continuance disappear more or less rapidly (Townsend, in a case accompanied by right hemiplegia and uræmia, saw these symptoms disappear after lasting for weeks); the amblyopia disappears, the vomiting ceases, the urine becomes more abundant, and the patient regains more or less appetite. Usually there is no return of the urgent symptoms, and as the secretion of urine increases the anasarca and serous effusions disappear, the brain and lungs are relieved, the appetite and the temperature of the body become normal. Under such circumstances the last symptoms to disappear are those on the part of the urine, which, however, become every day clearer, more free from albumen, and less turbid. After a while the albuminuria disappears entirely, and there remains only a scanty grayish white sediment, containing a few hyaline casts and a small amount of epithelium, to remind us of the past serious lesions of the kidney. Finally, even these vanish, and complete convalescence is established. For the first time there now takes place an intense general desquamation.

The form of scarlatinal nephritis which is characterized by general anasarca and dropsical effusions lasts, in favorable cases, from one to several months; recovery follows immediately, or soon after the disappearance of the dropsy. Occasionally the nephritis becomes chronic, or months after the apparent recovery a relapse occurs, with albuminuria or dropsy after some new exciting cause. Death often ensues even in the first, generally in the second or third, occasionally in the sixth week of the dropsy, or even later, partly as a direct result of the nephritis and partly as a result of other affections.

With the disappearance of the dropsy the series of scarlatinal symptoms generally ends. The normal course of the disease, when the kidneys are not involved, lasts from one to two weeks, the desquamation being generally completed at the end of the third week; but when nephritis with dropsy occurs, the affection may easily continue for six or eight weeks and in severe cases much longer.

Scarlatina, when it runs an irregular course from the start, presents such a great variety of symptoms, that it is impossible to give a general description which will answer for the several forms of the disease. The following, therefore, claims to be of service only as a general sketch.

Those cases which are *irregular from the start* are generally characterized by severe nervous symptoms with intense fever, and also by gastro-intestinal disturbance of unusual violence. After a period of incubation, in which there are no symptoms at all, or only a slight malaise, the attack is ushered in with intense headache and stupor, or with faintness, profound malaise, and repeated copious vomiting of a mucous or bilious character. Then rapidly follow more or less prolonged and severe convulsions, delirium, and a marked elevation of temperature. These symptoms may doubtless be present in normal cases, but never with the same extraordinary severity. The pulse very soon exhibits a rapidity which is seldom seen in other diseases; it is often intermittent or irregular, but never abnormally slow; at the same time it is generally full and hard, but, in rare instances, small. The skin, after the period of prostration, is turgescent and hot, the face bright red with actively pulsating arteries, the eyes are injected, and the patient complains of the light. There may also be other symptoms, such as increased dyspnœa, palpitation, tinnitus aurium, giddiness, grinding of the teeth, uneasiness, and sleeplessness. The throat is seriously affected from the beginning of the disease, and the intense injection extends over the whole oral cavity; the throat is swollen, and even at this early period is covered with diphtheritic deposits. The tongue is bright red, and soon becomes dry, and there is intense thirst. Sooner or later, usually at the end of the first day, or at the beginning of the second, but sometimes one or two days later, and in rare instances after a prodromal fever lasting for weeks, the *eruption* makes its appearance. It is sometimes, especially when it appears early, intensely red and accompanied by much general swelling and tenderness of the skin with excessive itching; at other times it is of a more dusky red color with a tinge of violet, or it may be livid, and then does not entirely disappear under the pressure of the finger; in rarer cases it is abnormally pale, and then is usually very transient. When the eruption is very abundant the skin is often infiltrated and has the appearance of leather. Sometimes, instead of beginning upon the face and neck, it makes its first appearance upon the abdomen, back, and limbs, or suddenly breaks out over the whole body at the same time. Less frequently the face or other parts remain wholly or almost entirely free from the eruption, and the only situations affected to any considerable extent are one side of the body, or the upper or lower half of the body, or the extremities. Sometimes the eruption is still more scanty. Gläser, for example, describes a form of the eruption in which it appears as a broad ring around the neck, or at the same time as a semicircle or half-moon on the forehead, or as a circle surrounding each elbow-joint; Wildberg found it occurring only on the joints, Zehnder as red spots scattered over the body, and Geissler as a redness on the abdomen, simulating the erythema produced by a mustard plaster.

After the appearance of the eruption the fever continues with scarcely any abatement; the febrile reaction is so continuous that it is difficult to discover any morning remissions, or, at any rate, only such as are unimportant. During the next few days the eruption becomes still more intense, and miliaria make their appearance in various places, sometimes vesicles of larger size filled with a turbid, purulent, or ichorous bloody fluid, and even bullæ as large as a hazel-nut (scarlatina pustulosa and pemphigoides). At this time not unfrequently occur certain lesions which may be regarded as unmistakable irregularities of the eruption: small petechiæ, ecchymoses of larger size, elevations of the epidermis filled with blood resembling hemorrhagic miliary vesicles. These lesions are, however, frequently due merely to intense congestion of the skin and delicacy of its vessels, and are consequently of less serious import when the eruption is abundant than when it is scanty and pale. The *throat affection* also very frequently presents irregularities in its course. As a rule, we may consider the course of scarlatina as irregular whenever the throat affection is very severe; but it is, of course, difficult to place the limit beyond which the angina is to be regarded as abnormal. In these irregular cases the tonsils become so enlarged that they nearly or quite touch each other, and interfere seriously with deglutition; the arches of the palate are very œdematous and of a bright red color, and the throat is often covered in various places with pseudomembranous deposits; the mucous membrane is often discolored and livid, excoriations occur, which often terminate in ulcerations or even in gangrene, and may give rise to serious hemorrhages and considerable sloughing, for example, of almost the whole tonsil. The cervical lymphatic glands in all these cases become much enlarged, and form thick bunches along the lower jaw; not unfrequently the entire cellular tissue of the upper cervical region becomes so swollen and indurated in the form of a semicircular tumor that the inferior maxilla is almost anchylosed. The return of venous blood is thus obstructed, and the face presents an unsightly deformity, particularly in the parotid region. Respiration, speech, and swallowing become very difficult, the latter almost impossible. The severe inflammation of the throat often extends to the nasal mucous membrane, presenting either a suppurative or diphtheritic character, with a copious, acrid, almost corrosive discharge, which produces excoriations and ulcers both within the nares and at their orifices. Under these circumstances respiration is carried on almost exclusively through the mouth, which consequently becomes dry, chapped, and fuliginous. Epistaxis in considerable amount may also occur at this time, and severe disease of the auditory apparatus resulting in complete deafness may be rapidly developed. Serious disease of the eye may also result from extension of the morbid process through the lachrymal ducts to the conjunctiva. In severe cases the appetite entirely disappears, there is often considerable pain in the stomach, and the vomiting continues or returns upon slight provocation. There is also often a persistent diarrhœa, with greenish and perhaps bloody stools of a most offensive odor, indicating serious intestinal lesions, and accompanied by tenesmus and tormina. Schönlein describes very severe colicky pains occurring periodically in the umbilical region, and designates them neuralgia cœliaca. The kidneys also are often affected at this time in the irregular cases, the urine becomes more scanty, turbid, and albuminous, and contains sometimes only the products of a catarrhal, less frequently of a parenchymatous nephritis, sometimes also blood in large quantities. The further symptoms of kidney disease are not usually developed at this time, and if there be any slight swellings they are generally due to congestion of the skin. The *severe cerebral symptoms*, which are usually present in these cases, and which were formerly for a long time regarded as signs of disturbance in the secretion of urine, are now more properly attributed to the blood-poisoning, high fever, or to other lesions, such as hyperæmia of the brain with capillary extravasations, hydrocephalus, partial softening and beginning meningitis. According to Wunderlich, we may gain a more comprehensive idea of the great variety of these symptoms if we compare them with other groups of symptoms depending upon definite lesions to which they present a striking resemblance. Not unfrequently the patient becomes suddenly paralyzed, as in apoplexy, and dies in a few hours, or even sooner. In such cases the paralysis rarely takes the form of hemiplegia, but generally affects the whole body, and is accompanied by a rapidly developed and profound coma with dilated pupils. In many cases the symptoms are those of severe cerebral irritation, either with delirium of a garrulous or furious character, or with epileptiform or tetanic convulsions, or trismus. These attacks may cease, or, more commonly, end in death, with coma, collapse, and paralysis, or they may even change their character. Sometimes these symptoms of irritation are intermittent with irregularly occurring paroxysms; during the remission the amount of cerebral disturbance may be very slight, or the affection may assume an entirely different form. The sudden and total change which takes place in such cases, after a longer or shorter period of repose, is often very striking. Wunderlich states that not unfrequently symptoms occur which resemble profound toxicæmia or a severe typhus fever, apoplexy (hemiplegia and paraplegia), encephalitis, meningitis, very acute or subacute hydrocephalus, and which are accompanied by retardation of the pulse and by the so-called Cheyne-Stokes respiratory symptom; much more uncommon are various local disturbances of sensation (paræsthesia, sleeping of the feet, hyperæsthesia, and anæsthesia), neuralgiæ, especially of the upper half of the body, local spasms (contractions of the neck, torticollis, spasm of the glottis, retention of urine, nystagmus, reflex cramps of various kinds), and local paralyses, such as aphasia and amblyopia, or paraplegia of the lower extremities, or, in addition to the latter, retention of urine and fæces, rigidity of the back, difficult deglutition, etc. In some cases, in connection with the severe and fatal cerebral symptoms, Paul found sugar in the urine. The form of the pathological changes corresponds only imperfectly with the symptoms, and it is never possible to diagnosticate from the latter the exact nature of the anatomical lesion. Sometimes, even when the cerebral and spinal symptoms are most severe, no abnormal change can be detected. Very rarely (at least such cases are rare in Leipzig), even during the first few days of an attack of scarlet fever, severe *rheumatic* affections may arise, especially inflammation of the joints with rapid progress to suppuration, and abscesses may form between the muscles, in the internal organs, etc. Sometimes severe pains are felt in certain parts, spontaneously or on pressure, either over the bones, as, for example, the spinous processes, the anterior surface of the tibia, the acromion, etc., or in the muscles when the patient voluntarily makes much muscular exertion (Betz). These pains are due to affections of the *periosteum* (perhaps also of the bones) and *muscles*, but not to any lesion in the joints. Heim, Joel, Betz, Stiebel, and others have called attention to a peculiar *odor* which sometimes emanates from scarlet-fever patients, and reminds one of the odor of old cheese or of a menagerie. It is so penetrating that it fills the whole room, and may be regarded as a pathognomonic symptom; but whether it is present only in the unmistakably irregular cases I am unable to say.

The symptoms above described do not always appear in equal intensity at the same time, but in numerous instances those of some particular organ assume predominance, while the others remain in the background or may be entirely absent. This fact furnishes a basis of distinction between a number of the chief varieties of irregular scarlet fever, which derive their names from the nature and intensity of their most important symptoms; *e.g.*, angina maligna, angina parotidea, scarlatina with severe nervous, meningeal, or apoplectic symptoms, or with rheumatoid or choleraic complications, etc. In all these various forms the eruption may vary extremely, in the time of its occurrence, in its amount, intensity, and duration; and this fact, together with the extraordinarily great number of the irregular symptoms and their combinations, accounts for the striking differences which are noticed in individual cases.

The severe forms of irregular scarlatina sometimes last only a short time, a few days, a week, or a little longer; when the local symptoms have become fully developed the farther course of the disease is often cut short by death, which may occur in various ways. Sometimes the fatal result is due to the severity of the fever, which is independent of any intense local symptoms, and which gradually or suddenly, or even after an antecedent remission, produces a very intense, and conseqently fatal elevation of temperature (107.6°-109.2° and upwards), and an extremely rapid pulse (200, and even higher). At other times death results from the angina and the disease of the cervical lymphatic glands, through obstruction of the air-passages, either by extension of diphtheria to the larynx or by œdema glottidis. Among other causes of death may be mentioned congestion of the cranial viscera, intense fever, pyæmia, septicæmia, and other constitutional affections, cerebral exhaustion from intense excitement, pathological lesions in the brain or its membranes (inflammations, hemorrhages, hydrocephalus, etc., or the rapid increase of a chronic hydrocephalus), tetanus or convulsions occurring unexpectedly with a premature and sudden disappearance of the eruption, rapid collapse with abrupt fall of temperature, various complications, and the hemorrhagic diathesis.

In favorable cases, after remaining well marked for a few days, a week, or perhaps longer, the eruption fades, the miliary vesicles dry up, and desquamation commences. Irregularities in the eruption may, however, occur at this time in the form of a more or less characteristic return of the scarlatinal redness, or in the form of other cutaneous lesions, such as roseolæ, erythema simplex and nodosum, erysipelas, urticaria, herpes, lichen, ecthyma, furuncles, etc., as an immediate result of the intense scarlatinal dermatitis. These symptoms are not dangerous, but merely protract the convalescence. The angina may also now return, unless it have already advanced to its more serious lesions, diphtheria, gangrene, etc., in which case the fever may continue for some time longer, and delay recovery in various ways. The throat affection may be still farther prolonged by the occurrence of severe glandular affections or intense inflammation of the cervical connective tissue, leading to suppuration and gangrene, or even to alarming hemorrhages from erosions of large vessels. These hemorrhages may also occur after the incisions of an abscess. Recovery may be delayed in rare instances by disease of the joints or kidneys, or by various other complications. The cerebral symptoms, as a rule, abate along with the fever. If the fever be not maintained at a high point by the excessive severity of the symptoms above described, or by the occurrence of farther serious complications, it gradually subsides, but more slowly and with less regularity than in the normal course of the disease; so that in an attack, which has been irregular from the start, but which is still benign in character, the normal condition may be re-established by the end of two or three weeks. Parenchymatous nephritis may, and often does occur at this time, with a return of the fever and other symptoms, and even in its mildest form may delay the convalescence for a considerable time. In such cases the course of the renal affection is often of moderate severity, but more frequently it is severe and attended by dropsy of the connective tissue and serous membranes, together with copious hemorrhages in the renal tubules.

According to this description the chief characteristics of many of the irregular cases of scarlet fever are the severity of the local lesions, the cerebral symptoms, and the intensity of the initial fever. But not unfrequently there occurs still another form of the disease in which there are not only local affections of moderate, perhaps even trifling importance, but also a disproportionately severe fever of long duration, which characterizes this variety as a *typhoid scarlatina.* In such cases the fever is the chief symptom, and the local lesion seems to be of subordinate importance, and yet sometimes the prolongation of the fever into the fourth week, or longer, is apparently wholly due to a protracted local affection. The eruption in this form of scarlet fever generally makes its appearance only after a rapidly developing initial stage, followed by high fever lasting for several days, and is then often very scanty, but otherwise normal. The angina, which is the next most important symptom, is often imperfectly developed, but appears somewhat earlier than the eruption. The brain symptoms and the fever are, however, quite severe, especiall yduring the first few days of the disease; the patient complains of headache, is delirious or drowsy, the facial expression much altered; there is urgent thirst, the tongue tends to become dry, but cleans off in the characteristic manner; there is some cough, and the skin, which desquamates in the usual way after the fading of the eruption, remains dry and hot. At a later period the patient is apathetic and very deaf, although the disease of the auditory apparatus appears to be but slight; the spleen enlarges, hypostatic congestion occurs in the lungs, and there is diarrhœa. The fever continues for weeks in a remittent form, but there is generally no other local affection except a moderate enlargement of the cervical lymphatic glands; in the third week signs of renal disease may make their appearance in the urine for the first time, and the fever is explained by the occurrence of dropsical symptoms. When the latter occur, the fever, which has hitherto been regular, now generally changes its character, increasing in an unexpected manner, and pursuing a course determined by the new local affection, or the febrile symptoms may abruptly cease entirely; less frequently when the renal symptoms are moderate, or altogether absent, the daily exacerbations diminish in severity and the fever gradually disappears. At the autopsy, when death occurs in this typhoid form at a late period, besides the usual affections of the throat, lymphatic glands, skin, and perhaps kidneys, there may be found also other lesions, such as hyperæmia of the brain, slight cloudiness of the meninges (sometimes with granulations, according to Hebra), catarrh of the respiratory passages, hypostases of the lungs, effusions into the serous cavities, engorgement and enlargement of the liver and spleen, marked disease of the follicles of the small intestine and infiltration of the adjacent mesenteric glands, sometimes even a slight typhoid tumefaction of Peyer's patches. This typhoid form of scarlatina occurs without any connection with typhoid fever, and we may dismiss, therefore, without any further consideration, the conjecture that this special course of the disease is due to such a complication.

Sometimes after an attack of scarlet fever which runs a typhoid course without special local symptoms, or is attended by various local diseases, or perhaps is uninterrupted by any complications, a fresh eruption unexpectedly breaks out over the whole body. In the cases which have come under my own observation, it is never exactly similar to the normal first scarlatinal rash, and yet the resemblance is so strong, and the difference from all other exanthemata so marked, that I have no hesitation in regarding it as an irregular second scarlatinal eruption, and shall therefore distinguish it from the true relapse, which occurs after the normal course of the disease, by calling it a pseudo-relapse. The cause of this anomalous eruption may probably be found in a renewed determination to the skin occurring at an unusual time. Nephritis appears to have no influence in producing the pseudo-relapse, at least this is the experience of myself and others.

The pseudo-relapse presents some notable peculiarities. It is decidedly more like roseola, and has not the finely punctate appearance of the first eruption. The roseolæ, which are very numerous, are generally of a dark, rather scarlet-red color, at the same time smaller and less sharply defined, generally closer together, and less elevated above the surrounding pale skin than is the case in measles. The eruption spreads over the body in a perfectly normal manner. Sometimes the roseolæ are developed everywhere quite uniformly and simultaneously, sometimes they appear only on the face and extremities or upon the latter alone, and at other times they are confined to the trunk, or even to certain portions of it. On the face the eruption affects also the region around the mouth, and there is no such sharp contrast shown by this region with the neighboring parts as is seen in the first attack even when the eruption is very copious. Very commonly this second eruption is completely confluent on certain parts of the body, at other times it is markedly discrete. If confluent, it is much more pronounced than in the first attack, for the individual roseolæ can no longer be distinguished from each other ; in some cases, besides the confluent portions, there may be present sharply circumscribed roseolæ, with no tendency to confluence. As a distinction from measles, the confluence is found not so much on the face as on certain other parts, such as the scalp, very often also the extremities, and particularly flexion-surfaces. The roseolæ do not reach their full development in all parts at nearly the same time, as in the first attack and in measles; but at one place they rapidly become confluent as soon as they make their appearance, in others they perhaps begin to form for the first time on the second day, remain of a pale color, and do not coalesce at all. The full development may take place at all points within twelve hours, or the eruption may appear gradually and incompletely only after the lapse of several days. When this second eruption fades it may disappear so slowly and with such interruptions during the febrile exacerbations that the process of decline may be mistaken for a maximum development of variable severity which has been protracted for several days. But in other cases an intense confluent eruption may arise and fade within little more than twenty-four hours. The desquamation following the fading of this eruption is always lamellar, generally copious, and follows immediately, even in cases where there has been little or no desquamation preceding the second eruption. The chief characteristics of the pseudo-relapse, such as the color, the commingling and often actual confluence of the patches, the unequal development on various parts of the body, the simultaneity of the eruption, the rapid development to a partial or general maximum, the manner of fading, and the intense lamellar desquamation—all these resemble the characters of the normal eruption, and yet they may be distinguished by the distinctness of the roseolæ and the intensity of the eruption on the face, including the region about the mouth. For these reasons I do not hesitate to regard the eruption as an irregular form of scarlet fever, and not an accidental complication. Other kinds of eruption, particularly miliaria, are, as a rule, absent.

During the continuance of the second exanthem the course of the body-temperature is by no means regular and characteristic. This fact may perhaps by itself enable us to distinguish the nature of the new process. I have noticed particularly that the maximum of the pseudo-relapse never coincides with the maximum of the temperature. According to certain observations, however, the new eruption appears to have a slight influence in elevating the body-temperature. There is always a moderate amount of renewed injection of the mucous membrane of the throat and parts of the mouth, the tongue also presents the same appearance as in the first eruption (redness, enlargement of the papillæ), and the cervical lymphatic glands undergo a moderate increase in size. Slight conjunctivitis, nasal catarrh, hoarseness, cough, and the symptoms of a slight bronchial catarrh may sometimes be noticed even before the breaking out of the second eruption. The mucous membrane of the alimentary canal is not much deranged, the liver is of normal size, but the spleen generally remains moderately enlarged. The nervous symptoms, if any be present, may be considered as the result of the febrile condition.

In general terms it may be said that the course of the scarlet fever is not much altered for the worse, at least not in any striking degree, by this very irregular outbreak of a second eruption; most of the cases recover. The cause of this abnormality is unknown; but the regularity of the interval which intervenes between the first and the second eruption makes it highly probable that there is a close relation between them. Perhaps the explanation lies in a peculiar phase of the development of the scarlatinal poison

Among the irregular varieties of scarlet fever the *hemorrhagic* form also deserves a special notice. As a rule, the condition of the patient is alarming from the start, and is characterized by intense fever and cerebral symptoms. The eruption is generally but very imperfectly developed; spots as large as a millet or hemp seed, with a clearly marked margin, and of a crimson or dark purple red color, and finally ecchymoses of larger size, make their appearance all over the body; the skin, which is at first erythematous, subsequently becomes flabby and wrinkled, and the face bloated. In all the more severe cases there is considerable angina, sometimes of a diphtheritic or gangrenous character, the lymphatic glands and connective tissue of the neck usually present serious lesions, and when gangrene or diphtheria occurs the result is almost always fatal. Hemorrhages frequently take place in the cavities of the body or from the mucous membranes, especially the latter, if, as is often the case, they are much diseased or ulcerated. Hemorrhages occur most frequently from the mucous membrane of the nose and colon, and may produce the most profound exhaustion. Severe hemorrhage may occur also from a tooth-cavity, or from slight wounds such as leech-bites, or the scarification for cupping. It may also take place from the urinary passages, usually from the substance of the kidney, and in rare cases from the pelvis or the lower portions of the urinary passages. In these cases the urine is of an almost blackish color. In women hemorrhages occur from the genital organs, either with or without the presence of vaginal diphtheria. The internal organs, especially the brain, heart, liver, spleen, stomach, and lungs, may also be the seat of hemorrhage; so also the subcutaneous cellular tissue, especially of the neck. Finally, pleuritic or pericardial exudations may occur of a more or less hemorrhagic character. In the hemorrhagic form of scarlatina death is by far the most frequent termination, and sometimes depends upon the intense fever; sometimes upon prostration from hemorrhages and diarrhœa; sometimes upon cardiac exhaustion or serious organic affections, such as pneumonia, meningitis, nephritis, colitis, pleuritis, etc., and finally, upon the severity of the constitutional disturbance, either directly from the scarlatinal poisoning or from secondary affections, such as diphtheria, gangrene, etc. Recovery is possible only in the milder grades of this irregular form of the disease, where the fever is slight, the angina still more unimportant, the hemorrhages trifling or of brief duration, and when no other serious complications occur.

Irregularities in the course of scarlet fever may occur at any time in consequence of the development of *complications*. If, as a matter of experience, the latter are usually found to have but a slight effect upon the progress of the disease, the explanation may be found in the fact that the irregularities precede and are not caused by the complications.

It is easy to see what extraordinarily varied symptoms may be produced by the combinations of all these numerous symptoms, and by the different general types which the course of the disease presents: in fact, no other disease exhibits such a variety of forms, such a dissimilarity of symptoms in cases which depend upon a common cause.

When scarlatina occurs in the course of other diseases (which are then generally irregular) it frequently increases their severity: according to Rilliet, especially if the other affection implicate organs which are apt to be affected by the scarlatinal poison. Thus, for example, Löschner regards the existence of a previous tuberculosis of the lymphatic glands as a very bad sign in scarlet fever: the more severe and extensive this lesion is, the more violent is the course of the disease, the more frequent its complications (meningitis, hydrocephalus, pleuritis, dropsy), and the more unfavorable its termination. On the other hand, other diseases which are indifferent to the scarlatinal poison not only do not change the character of the scarlet fever, or interfere with recovery, but they may even exercise a salutary influence. Barthez and Rilliet state that scarlatina has a favorable influence upon phthisis, an observation which is approved by Deiters, but rejected by Hebra. In this category is included the concurrence of scarlet fever with small-pox, measles, varicella, typhus, etc. In these cases death results only very rarely. In two cases of chorea seen by Thompson, the disease disappeared when the children were attacked with scarlatina: Löschner saw two cases, one of which recovered during scarlatina: in the other the disease was aggravated but suddenly disappeared. Lumbricoid worms are frequently discharged during the course of scarlet fever, and cases have been known in which the worms have passed into the stomach and been vomited. Prevot has seen scrofula in a child cured by the occurrence of scarlatina maligna with formation of abscesses. The child afterwards permanently regained vigorous health. Betz observed an improvement in the stools in a case of dysentery when a diphtheria of the colon occurred as one of the symptoms of a complicating scarlatina. The child died.

Complications.

The *complications* of scarlatina are very numerous, and at the same time of a very varied character; there is scarcely any organ which may not be implicated. No period of the disease is free from complications: they may occur during the initial stage, the fastigium, or the decline; some at any time, others more particularly at certain periods; in normal cases, or in those which are irregular from the outset, but more especially the latter; hence they are more frequently observed in severe epidemics than in those of a milder character. They occur also in connection with every form of development of the eruption, angina, or fever; whether the eruption be intense, or moderate, or imperfectly developed, or entirely absent, or whether it run a brief and rapid or a protracted course. Nor does the form of the angina or the grade of the fever appear to have any influence in their production.

The forms of the development of these complications is extremely various. Sometimes they occur suddenly and most unexpectedly, or they may be preceded by certain insignificant and scarcely noticeable symptoms, without any external cause or other disturbance, or they may perhaps seem to depend upon the sudden disappearance of the eruption. In the latter case there will always be a doubt whether the complication be not due rather to some new internal morbid process. The complications may progress more or less rapidly, and sometimes several of them may combine in the most varied manner.

In many cases the occurrence of an important complication is the immediate cause of death; in others it merely protracts to a greater or less degree the course of the disease, or gives rise to sequelæ.

The most frequent complication of scarlet fever is that with severe cerebral and spinal symptoms, and this for the reason that the latter always occur whenever the disease runs a severe course, or whenever there are other serious complications. And yet in many instances it may be a question as to what is the true nature of these symptoms, since it is not easy to distinguish between a merely concomitant symptom of an uncomplicated case and an actual complication. A positive discrimination is, however, without much practical importance.

Severe nervous symptoms can be traced back to several causes: to the scarlet-fever poison in the blood acting as a narcotic; to the high fever; to the extensive areolar infiltrations in the neck, which interfere with the return circulation of the blood; and in the latter stages of the disease to the anæmia produced by the severity of the fever; to the blood alterations from pyæmia and septicæmia, and finally to the changes produced in the course of the so-called uræmia. The anatomical alterations, which are not, however, invariably found on examination, are hyperæmia, slight inflammations, œdema, anæmia, and hydrocephalus—rarely severe inflammation with abscesses and extravasations. In some cases it is impossible, either at the outset or even in the whole course of the disease, to trace the cause of the symptoms. The symptoms themselves do not give us the slightest, or at least only the most dubious indications of the nature of the nerve lesions, although an experienced judgment is of great service in their detection. The difficulty in the diagnosis becomes still more apparent when we consider that each symptom may depend upon a great variety of lesions. The nervous disturbances appear at various periods of the disease: at the outset, and then generally in connection with other symptoms, as an indication of toxicæmia and of the severity of the fever (headache, delirium, and coma); perhaps suddenly, in the midst of an otherwise favorably progressing case, brain symptoms may arise, as a result of cerebral irritation; also when the eruption, fever, and angina are rapidly increasing in intensity, in the form of febrile delirium and stupor, increased or modified by the continuance of the poisoning; also coincidently with the sudden disappearance of the rash, and then especially in the form of convulsions during the stage of nephritis and uræmia, in the same way as when there is no scarlatina; in fact, at any time whenever pathological changes occur in the nervous system.

Affections of the eye of a mild character, particularly slight conjunctivitis, with epiphora and photophobia, are not infrequently met with at the outset of severe scarlatina, coincident with an abundant eruption on the face, but never to the same degree as in measles. On the other hand, the later occurring ophthalmias, which are not infrequently met with in scrofulous subjects, are generally very obstinate, and lead often to marked corneal troubles and even panophthalmitis, with severe and even permanent impairment of vision. The same symptoms may result from an intense coryza passing up through the lachrymal duct, and thus giving rise to lachrymal fistula and other more or less severe affections of the tear-passages. Primary keratitis occasions frequently perforation of the cornea, with its unfortunate consequences. From the scarlatinal nephritis a special form of retinitis arises, which, according to Schröter, develops itself sometimes within a few days, sometimes in a longer time, with various degrees of loss of sight. It occurs generally in both eyes, but not always at the same time nor in the same degree, and, as a rule, total blindness is not developed. The course of the affection is generally protracted, but the prognosis is generally favorable, more or less complete restoration of vision being the usual result. Less frequently the impairment of vision is due either to atrophy of the optic nerve or to a detachment of the retina.

The transitory blindness which accompanies uræmia, in connection with other symptoms, is of a far different nature, and occurs independently of the presence or absence of any retinal affection. It shows itself, Schröter says, as a binocular amblyopia, which proceeds in a day or two to complete loss of perception of light—not unfrequently on awakening from a period of unconsciousness the patient finds himself completely blind. The pupils are abnormally dilated, they usually still react to light, but less frequently are fixed and motionless. The amaurosis disappears as rapidly as it occurred, and generally in from two to four days vision is completely restored. Porter has observed in a girl six years of age such a temporary amaurosis, with exophthalmos from infiltration of the cellular tissue of the orbit, with other severe complications. Duval, under similar circumstances, found the sight completely restored after the exophthalmos had lasted for ten days.

The previously described inflammatory *ear affections*, or a relapse of the same if any occur, often begin, according to Wendt, with very violent pain in the ear, and a febrile exacerbation which is independent of the progress of the eruption, etc. The hearing is always very much impaired by the swelling of the mucous membrane, also by the closure of the Eustachian tube, by the filling up of the tympanic cavity with hypersecretion, and by destructions of moderate extent. Subjective tinnitus is seldom met with. Sometimes brain symptoms intervene through the free anastomoses between the dura mater and the middle ear by means of the middle meningeal artery. Hynes saw a boy die on the twenty-second day of the fever from arterial hemorrhage from the right ear, and at the same time from the mouth.

The affection of the *nasal mucous membrane* (coryza scarlatinosa) begins, as a rule, at the time of the acute angina, less frequently in a subacute and simple form, and more often as a severer difficulty, with great swelling and intense pharyngitis and diphtheria of the naso-pharynx. It is indicated by the nasal tone of the speech, but in particular by the more or less abundant irritating ichorous discharge from the nostrils, with occasional epistaxis from the intensely congested mucous membrane, or at times from ulcerated surfaces. The scarlatinal coryza is truly a complication very much to be dreaded, not so much on its own account, as on account of the dangerous forms of pharyngitis associated with it. It may, however, of itself give rise to quite grave lesions, especially through the formation of ulcerations, which, in their progress, may excite serious hemorrhages, and may even lead to periostitis and necrosis of the nasal bones.

Under similar circumstances we find, principally in young children, severe *stomatitis* appearing, with ulceration affecting the back part of the mouth, the cheeks, gums, and tongue, and spreading thence over a considerable portion of the mucous membrane of the mouth. This affection is painful, gives rise to a very offensive odor, salivation, and hemorrhages, and interferes seriously with the nutrition of the patient.

Pure and simple *ptyalism*, without parotid or other affections of the mouth, is seldom seen (Müller); it generally occurs in connection with ulceration of the mouth, and probably from the use or abuse of mercury. Zehnder saw it return once after a cessation of fourteen days caused by uræmia.

The inflammation of the whole or at least the greater part of the *connective tissue of the neck*, as well as of the *parotid region*, which usually occurs in the second week of the disease, but sometimes earlier, is correctly regarded as an extremely perilous affection, not only on account of the great danger of suffocation from compression of the air-passages and œdema of the glottis, but also and principally on account of the extensive destruction of the tissues resulting from the great suppuration, or from sloughing. From the suppuration we can have deep burrowing as far down as the thorax, perforation of the pharynx, so that the food comes out through the external opening (Cremen), retro-pharyngeal abscess, erosion of the large veins of the neck, as well as of the carotid and smaller arteries (arteria lingualis), and consequent fatal hemorrhage, and lastly, destruction of important nerves and muscles. Gangrene produces still more readily the same consequences, and besides these, emphysema of the skin. By the destruction of the skin the deeper parts are laid bare to a large extent, and septic infection often results. With any extended destruction of the tissues death is almost inevitable; it ensues sometimes quickly, sometimes more slowly from progressive marasmus.

Affections of the *air-passages* and *lungs* are much more rare in scarlet fever than in measles. The most important are: œdema of the glottis from severe sore throat, inflammation of the cellular tissue of the neck, with or without ulceration of the larynx; croupous laryngitis and similar disease lower down in the air-passages, with or without broncho-pneumonia, resulting from pharyngeal diphtheria; bronchitis, which sometimes occurs as a complication during the height of the eruption, or immediately after it; under similar circumstances, and yet more rarely, croupous pneumonia of greater or less extent, affecting the upper as well as the lower lobe of the lungs; this affection does not make the prognosis more unfavorable in the absence of other complications; pneumonia, lobar as well as lobular, very fatal in the later stages of scarlatinal nephritis, because it is often conjoined with pleurisy, and further on gives rise to pulmonary œdema and abscess of the lung; it is usually attended by signs of great constitutional disturbance, such as pain, dyspnœa, distressing cough, abundant frothy, bloody sputa, and augmentation of fever; atelectasis of the lungs in connection with other severer symptoms; pulmonary apoplexy, especially when a hemorrhagic diathesis is present. Fleischmann had a case of considerable hemorrhage into the posterior mediastinum from a cavity of the lung; in rare cases gangrene of the lung occurs; finally, there may be œdema of the lungs, which may unexpectedly occur in every stage of the disease, with a fatal issue, and when occurring at an early period of the fever is generally due to sudden failure of the heart's action from overstrain of that organ.

Symptoms sometimes appear, such as severe gastralgia, persistent and often bilious vomiting, and great sensitiveness of the stomach to all ingesta, denoting a *gastric affection*, which, though intense, is free from danger and of short duration. In cases of hemorrhagic scarlet fever hæmatemesis sometimes occurs.

The follicular inflammations and partial or general hyperæmia of the *intestinal mucous membrane*, occurring at the time when the eruption has attained its fullest development, manifest themselves by a persistent and considerable diarrhœa, with or without colicky pains and tympanites, and may perhaps lead us to mistake the disease for a typhoid fever. At the same time the tongue is dry, there is considerable thirst; the urine is scanty, the eyeballs are deeply sunken, and the face has the expression of a patient suffering from cholera; the stools are liquid, mucous, yellowish or greenish, even dark green, contain only a small quantity of liquid fæcal matter, but a large amount of epithelial cells and pus-corpuscles, sometimes also more or less blood. If these symptoms are only of slight intensity, they do not denote any danger; but if they are more severe they are rapidly followed by collapse. Joël observed a case where, besides the intense fever, the gastro-intestinal symptoms were the most important phenomena. The slight angina, the subsequent desquamation of the epidermis at different parts of the body, and the fact that the sister was also suffering from scarlet fever, did not leave any doubt about the diagnosis in this case. Enterocolitis and coloproctitis, with violent colic, painful tenesmus, and catarrhal or dysenteric evacuations, are occasionally observed during the course of scarlet fever, especially in its later stages. If sporadic, they generally last only a short time, and are not particularly dangerous.

At the time when the eruption is at its height (and even later, according to Stäger) the *liver* is sometimes found considerably enlarged, in consequence of congestion; sometimes it is smaller than natural. At the autopsy of an adult dying from scarlatina, Wunderlich found the liver weighing but little more than two pounds. Icterus may occur as a result of the slowly progressing changes in the liver, but becomes marked only after the disappearance of the eruption. Santlus states that he has frequently observed icterus as a complication of scarlet fever in an epidemic distinguished by numerous anomalous cases. Patients very frequently recover from this complication (Swiney, Graves, etc.), but a fatal termination is not rare. Danielssen observed a case of a girl, twenty years of age, who died with symptoms of malignant icterus. The autopsy showed acute fatty degeneration of the liver, spleen, kidneys, heart, and considerable ecchymosis of the lungs and kidneys. But, as a rule, hepatic lesions are rare.

Whenever a careful examination is made, especially during the eruptive stage, an enlargement of the *spleen* is frequently, if not almost constantly found. It must be regarded as a complication only in those rare cases where the spleen is unusually enlarged and softened; the symptoms produced by it are not characteristic.

Inflammations of *serous membranes* occur generally in connection with scarlatinal rheumatism of the joints. Meningitis forms the principal exception to this rule, and occurs most frequently at the beginning of scarlet fever, at the time of the severe cerebral congestion. It is marked by a stage of extreme irritation (most intense headache, glistening eyes, loud screaming, boring of the head into the pillow, vomiting, high fever), with early signs of compression of the brain (sopor, convulsions). Those cases of pleuritis caused by some pulmonary affection also form exceptions. All forms of scarlatinal *pleuritis* are characterized by rapid development and by but slight local disturbance, even when the affection is very intense. From some unknown cause, the effusion is generally unilateral and sometimes purulent, as is often the case in many other exudations of scarlet fever. The *pericardial effusion* possesses these peculiarities in a much lower degree. It occurs more rarely, is generally moderate in amount, gives rise usually to but slight subjective symptoms, and is sometimes further complicated by *endocarditis*, which may be followed by its usual sequelæ, chronic valvular disease, and embolism of peripheral arteries resulting in gangrene, pyæmia, and septicæmia. Endocarditis may occur as an isolated complication of scarlet fever, or as a result of the rheumatic affection. *Peritonitis* is characterized by either a turbid or a more or less abundant purulent exudation, and adhesions of the intestines at numerous points. According to Hebra, these lesions are usually the result of tuberculosis of the peritoneum or mesenteric glands ; and here, as in the ordinary form, there is very severe pain, tympanites, and paralysis of the abdominal muscles. I have often found similar symptoms present in cases of commencing nephritis, and thought that this disease was associated with peritonitis.

Purulent arthritis is a rare complication. The joint seems to be only slightly affected during the first days, but subsequently it becomes more painful, red, and swollen, the fever increases, the patient becomes delirious and suffers from other grave adynamic nervous symptoms, finally death closes the scene. Or the inflammation is intense from the beginning, and the complex symptoms of pyæmia rapidly manifest themselves, with extension of the arthritis to one or more additional joints. In rare cases the purulent arthritis terminates in recovery after the formation and cure of tedious fistulæ. When the patient is scrofulous or exhibits some other allied diathesis, granular synovitis with chronic suppuration (fungous arthritis) generally occurs in place of the simple affection.

As complications of scarlatina, which occasionally occur at any period of the disease, the following affections are worthy of mention : inflammation of the sheaths of the tendons, which is very apt to be suppurative inflammation ; inflammation of the muscles ; extensive abscesses in the muscles of the extremities, trunk, or cervical region, also elsewhere, as in the mamma of a girl fourteen years of age, as reported by Weber, or extending from the genitals and forming fistulous openings into the hip-joint, as in Wood's case ; periostitis and ostitis in various places (the petrous portion of the temporal bone in case of otitis, the nasal bones in coryza, the cervical vertebræ in cases of severe pharyngitis).

Gangrene may involve other parts of the body besides the neck ; for instance, the genitals (Wood, Arrigoni) and perinæum ; the face, from diseases of the eyelids (Zehnder), the eye, ear, nose, etc.

Sequelæ.

Scarlet fever is sometimes followed, especially under unfavorable surrounding circumstances, by a state of anæmia, sickliness, and susceptibility to various slight affections, such as local œdemas, which are generally slight, but sometimes more extensive and recurrent. In Welsch's case, without nephritis, the œdema returned every eight days for twenty years, and sometimes even threatened life. The œdema is occasionally widely diffused and firmer than usual. The patient may also suffer from miliary eruptions, furuncles, abscesses, glandular enlargements, the milder forms of neuralgia, digestive disturbances with or without colic, just as after other grave diseases. Or there may be other derangements resulting from the various irregularities and complications which occur during the course of the disease. The following are the most important conditions following ulceration of the integument: lesions of the lymphatic glands, cellular tissue, bones and joints (intense articular pain, torticollis from inflammation of the cervical vertebræ, caries of the vertebræ with subsequent kyphosis, chronic purulent synovitis with formation of fistulæ, luxation, etc.), intestines (chronic diarrhœa, prolapsus ani in little children), the pharynx, air-passages, and genitals; sometimes also loss of hair, various cutaneous diseases, enlarged tonsils, chronic nephritis (which, according to H. Weber, may be found even in cases where no such disease existed during the scarlet fever. This is questionable, for nephritis scarlatinosa may run its course for a long time without producing any symptoms); hepatic and splenic affections, peritonitis, cardiac, especially valvular diseases, thrombosis of veins, stones in the bladder; likewise affections of the respiratory organs, such as chronic laryngitis following œdema glottidis, ulceration, etc., enlargement of the bronchial glands, pneumonia and pleuritis; miliary tuberculosis and phthisis. Of very great importance are the affections of organs of special sense. In many cases hearing is permanently impaired, in consequence of adhesions, rigidity, or even destruction of parts of the apparatus for conducting the sound; or we may have a sequel which not only endangers the hearing, but also the life, as, for instance, chronic suppuration of the middle ear, caries, etc. Less dangerous are the disturbances of vision, especially those which appear from two to six weeks after scarlet fever, from paralysis of the muscles of accommodation, and are always binocular and generally not connected with mydriasis. In these cases distant vision is good, except in the higher degrees of hypermetropia; the near vision is very poor, but can be perfectly corrected by strong convex glasses. These diseases of accommodation are generally sequelæ of diphtheria, as is shown by the existence of other signs of diphtheritic paralysis. They may cause convergent strabismus, which occurs only periodically in the beginning (during near vision), but ultimately becomes permanent. The external muscles of the eye are rarely paralyzed. Finally, there not infrequently occur nervous disturbances of various kinds: motor lesions, such as chorea after rheumatism with or without cardiac disease, hemiplegia, spinal and cerebral paraplegia, spasmodic contractions (of the trapezius and sternomastoids); paralysis of single nerves (the facial with or without disease of the petrous portion of the temporal bone, the nerves of special sense, etc.); further, general hyperæsthesia of the entire body (with the exception of the face and scalp), or hyperæsthesia and anæsthesia in various parts, different forms of neuralgia; hysteria and epilepsy; alalia, aphasia, and alexia; melancholia, mania, and other chronic mental derangements, especially in adults.

Diagnosis.

The diagnosis of a characteristic scarlatina eruption is not difficult. The minutely punctate appearance from the start and still noticeable even after the eruption has become confluent, the more or less uniform development of the eruption over the body, the absence of the eruption from the face and especially the region about the mouth, are all so characteristic that it is hardly possible to make a mistake. In difficult cases we ought to observe whether the individual spots are sharply defined and slightly infiltrated, or whether they shade off gradually and are smooth, as is frequently the case in primary erythema, especially in young children, in secondary erythema after wounds and operations, in the suppurative stage of variola, and in eruptions produced by the application of tar or creosote. Erythema is the only eruption which presents a close resemblance to a perfectly developed scarlatinal rash. It may be distinguished, however, by the fact that it is not widely diffused, being absent on the extremities, neck, and portions of the trunk, and that it spreads in a very irregular manner.

Where doubt arises from the scantiness of the eruption, or from other causes, we must bear in mind that in scarlatina the angina is rarely absent (even in mild cases it is tolerably well marked); that the tongue almost always has the characteristic appearance, and that in most cases there is even at an early period more or less swelling of the cervical glands. In cases where an early diagnosis is impossible, the doubt will be cleared up at a later period by the occurrence of desquamation, particularly the characteristic desquamation on the palmar and plantar regions, or perhaps by the appearance of albuminuria with or without dropsy or other symptoms of nephritis. No other disease presents a group of symptoms similar to this.

In other cases where there is a suspicious angina, either with or without a scanty eruption, the diagnosis may be made from the appearance of the tongue, from a subsequent eruption, or from the occurrence of a cervical lymphadenitis or a nephritis. The scarlatinal poison may excite merely a trifling catarrhal angina, or perhaps only a follicular tonsillitis.

So also a rheumatic affection or a nephritis may be regarded as suspicious, if they occur in connection with the characteristic scarlatinal tongue, desquamation of the epidermis, and cervical glandular swellings. Equally suspicious is an angina accompanied by fever of a typhoid character, severe gastro-intestinal symptoms, or some other abnormal localization of the scarlatinal poison.

The diagnosis is more difficult when the scarlatinal rash is irregular, and presents a roseolar appearance. In such cases the affection is most apt to be confounded with measles. For the differential diagnosis, see page 106.

The distinction between rubeola (Rötheln, German measles) and scarlatina is still more difficult. Where the rubeolous angina is quite marked, and the lymphatic glands are enlarged, the rubeola may be easily mistaken for a slight attack of scarlatina with an irregular roseolous eruption, especially if there be no subsequent desquamation. We must then be guided by the short duration of the eruption, its presence on the face—where it appears as early and as characteristically as on the neck and trunk—by the scarlet tongue, by the form of the affection of the palate—in scarlatina only the posterior parts, uvula, arches of the palate, and their vicinity are affected, while in rubeola the anterior parts are also affected, and both to much the same degree—and finally by the fact that the glands are more seriously implicated in scarlatina.

The differential diagnosis between a slight scarlatinal rash, complicated by numerous sudamina, and a simple miliary eruption, is based upon the following: The latter is never so generally diffused over the whole body, as in the case of a scarlatina miliaris, whose papules will be found more distinct on the dorsal surfaces of the hands and feet, and which here and there will show a few characteristic places, with perhaps even confluent redness. If other symptoms of scarlatina, such as angina, the characteristic tongue, glandular enlargements, etc., be present, these will aid us in making the diagnosis.

Erysipelas may also be sometimes mistaken for an attack of scarlatina of limited extent. The distinction lies in the fact that in scarlatina, besides the other characteristic symptoms, the rash has a punctate appearance, while in erysipelas the surface of the eruption is not elevated, there is marked œdema of the connective tissue, and vesicles may form. In scarlatina, desquamation may occur in places where there has been no antecedent eruption; in erysipelas this is never the case.

Still more perplexing are the cases which occur without eruption, especially those rudimentary malignant cases which rapidly prove fatal. In such instances we must be guided by the fact that there is an epidemic of scarlatina at the time, or by the existence of more or less angina, or by the extremely rapid and intensely febrile course of the disease in connection with severe nervous symptoms which cannot be explained in any other way; in no other disease does death take place in so short a time and with such symptoms. The whole skin should be carefully and repeatedly examined in all such cases, inasmuch as the eruption is sometimes very transient, appearing perhaps only on the extremities or neck.

The scarlatinal nature of the dropsy, at whatever period of disease it may occur, can be recognized by its acute, almost cyclical course, which, in children especially, is not often seen from any other cause; perhaps also by the occurrence of desquamation, or by the retardation of the action of the heart; or by the fact that the œdema makes its appearance first upon the face, and changes its position from time to time; finally, by effusions into serous cavities, glandular enlargements, etc.

Prognosis.

The prognosis of scarlet fever is uncertain under all circumstances. Even when the disease is running a mild and perfectly normal course dangerous symptoms may arise, without special reason, thus changing at once the entire nature of the disease, and even very rapidly hastening death. The physician, therefore, should never predict, unconditionally, that the disease will end favorably, but he should always realize that unexpected contingences may possibly occur. Apart from such occurrences, which are far more frequent in the anomalous forms than in the normal, the prognosis is decidedly favorable; and is positively unfavorable only in some of the irregular forms. In forming a prognosis, therefore, the point of most importance is to distinguish whether the case is normal or abnormal, and if abnormal, whether the anomalies are of such a kind as to justify the hopes of a favorable issue or the reverse.

We may anticipate that scarlet fever will run a normal course under the following conditions: when the initial symptoms are mild; when the appearance of the exanthem is timely; when the outbreak is not sudden; when, in the main, it has a general distribution and completes its course rapidly, reaching its maximum early, that is, on the first or second day, or latest, at the beginning of the third; when the throat affection is inconsiderable, and there is neither great difficulty in swallowing, nor marked swelling, nor any other affection of the tonsils or cervical lymphatic glands; when the fever never exceeds 104° Fahr., and there is only moderate frequency of the pulse, and the cerebral symptoms at most are slight, and only last a short time at the above-mentioned height of the disease; and, finally, when the abatement in the eruption is accompanied by a steady decrease in the temperature, which after a few days returns to the normal; at least these conditions furnish some guarantee that there is no further localization of the scarlatinous poison. Should the disease, however, not end here, as is quite possible in the most typical cases, we may distinguish the following conditions as also normal and indicative of a favorable termination: slight affections of the joints; a nephritis, if it commences slowly, is of moderate intensity and of short duration, and if the fever and hæmaturia disappear about the third week, with only slight albuminuria, and little diminution in the quantity of the urine, as well as without cerebral symptoms and without dropsy; finally, an entire absence of any complication betokens a favorable issue.

Scarlet fever with an anomalous course is particularly unfavorable, and the prognosis is at least very uncertain, especially when the following conditions are present: a continuous rise in the temperature from 104° Fahr. upwards, or hyperpyrexia, with dyspnœa, and extreme frequency of the pulse, and also attacks resembling collapse, accompanied by a cold surface and small pulse; an exanthem of a very intense copper color extensively diffused, and lasting several days during its greatest height, or a livid eruption, or even, perhaps, one that has merely a trace of a blue color, particularly where there are abundant hemorrhages in the skin; angina gangrenosa, and diphtheria of the pharynx, especially when it extends to the nose, larynx, and air-passages; a very dry tongue; an intense coryza; great infiltration of the connective tissue in connection with lymphadenitis of the neck and periparotitis, chiefly when it terminates in gangrene, or by the discharge of matter inwardly, as into the œsophagus and air-passages, or by extensive burrowing of matter; typhoid forms; severe nervous symptoms of any kind, particularly paralysis of many muscles, deep coma, repeated spasms, eclamptic or purely tetanic spasm of the glottis, continuous delirium, and a maniacal condition, complete sleeplessness, the greatest restlessness and excitement; very frequent and long-continued vomiting, with abundant dysenteric diarrhœa, especially at the outset, the latter at a later period also, and then with or without frequent attacks of vomiting and severe colicky pains; purulent inflammation of the joints with or without pyæmia, with a general formation of abscesses; early commencement of nephritis, general dropsy with effusion into the serous cavities, excessive hæmaturia and albuminuria, as well as great diminution in the secretion of urine, or complete anuria for several days with or without the signs of uræmia, and with or without fever; all the more important complications, such as pleurisy and pneumonia, œdema of the glottis, hepatitis, endopericarditis, peritonitis, meningitis and hydrocephalus, keratomalacia, and gangrene of the eye; severe affections of the ear; the hemorrhagic diathesis; a condition of deep infection (septicæmic) resembling narcotism, without local manifestations, sometimes developing rapidly, especially at first, and sometimes slowly, and then generally at a later period.

I find, in addition, that the following noteworthy prognostic signs are cited, as being, to a certain extent, very unfavorable: Geissler, for instance, believes that a remarkable sprightliness and vivacity just before the attack bodes ill; Marcus thinks it a bad sign when there is a sharply defined redness of the face, with a chalk-white ring about the mouth and a tremulousness of the lower lip; Krauss fears the result when there is a white streak traversing the dusky-red or livid face; Reil has observed a chalk-white ring between the forehead and lips, while the nose appeared white and pointed; many of the older writers were exceedingly afraid of a sudden, or, at least, rapid abatement of the eruption; moreover the variableness of an eruption, which was now pale and now florid, now more fully developed at one point and now at another, was a cause for anxiety. On the other hand, the following symptoms are said to be favorable: a spontaneous hemorrhage from the nose when there are severe cerebral symptoms, excluding, of course, the hemorrhagic diathesis; a profuse salivation; and the occurrence of sudamina, if they be not too numerous.

Sporadic cases of scarlet fever are not always entirely benign; many authors believe, indeed, that they should be regarded as more dangerous than the epidemic forms. At any rate, sporadic cases are in the minority. The prognosis, in by far the greater number of cases, is chiefly determined by the character of the epidemic, and this term expresses the total result of the action of various factors of time and place in developing an average specimen of the disease: the character is good or bad, according to whether most of the epidemic cases have a normal course or an unfavorable or fatal one. But few epidemics are benign in character, and the majority of physicians now living will subscribe to the statement of Löschner, uttered more than thirty years ago, when he said, "I have never seen a benign epidemic," and they will lament that the time of Sydenham has passed when scarlet fever "vix nomen morbi merebatur." The scarlet fever of to-day owes its danger to the frequency of its anomalous forms. In this connection it is remarkable that the epidemics of various places and periods are apt, for unknown reasons, to be anomalous in single characteristics. For example, in one epidemic severe sore throat with inflammation of the cervical connective tissue may be a very marked characteristic, while in another diphtheria will be very common, and in a third there will be rheumatic affections, as in the Rhenish provinces and in France; in a fourth, again, there will be dysentery, while others again are extremely deadly from the frequency and severity of the dropsy. (In Senfft's cases 38 per cent. of the dropsical died.) Still, the great majority of the epidemics do not owe their danger to a single anomaly, but to several. For in almost every case there is deep infection, severe fever, complicated angina, severe cerebral symptoms, and extensive dropsies; while many persons who have successfully defied the fever, the nervous disturbances, and the angina and its consequences, finally die dropsical.

Richardson observed two very benign epidemics, without deaths, on two ships of war (comp. p. 177,); so, too, did Gillespie in 1852 (seventy patients); and so, according to Graves, whenever scarlet fever appeared in Dublin, between 1800 and 1834, it was always mild, and on one occasion, when eighty children were taken sick in an institution, there were no deaths. In 1834, however, there was an epidemic of extraordinary virulence and extent; in this case the character of the fever was far from being harmless.

Sporadic epidemics are not, however, always benign any more than are sporadic cases, as is shown by Palmer, when he describes the epidemic of Pleidelsheim, where the neighboring localities were spared. In this instance, out of 1,500 inhabitants, 201 were attacked, and there were forty-four deaths, so that there was a mortality of 21.9 per cent. Many patients died within the first twenty-four hours.

Epidemics of scarlet fever where the mortality is below 10 per cent. may be regarded as benign epidemics, comparatively speaking.

Such have been reported, as for example by Lee, where there were 309 patients with 13 deaths, a mortality of 4.2 per cent.; by Köstlin, of Stuttgart, in 1856, where 149 died, or 7.3 per cent.; by van Holsbeek of Brussels, in 1856, where about 8 per cent. died; by Kolb, of Amberg, in 1862–3, a mortality of 5 per cent.; by Cremen, where of 1,346 sick, 125 died, or 9.2 per cent.; by Gutherz, where of 76 sick, 3 died, or 4 per cent.; by Eulenberg, of Coblentz, in 1859, a mortality of 5 per cent.; by Höring, in two villages near Heilbronn, in 1856, 8 per cent. mortality.

The majority of the epidemics described lately have shown a much greater mortality, and yet it must not be assumed that the statistics which have been made known to us in most of the publications illustrate the average mortality of scarlet fever, for an epidemic has often been reported because it was remarkable for its virulence. The mortality very frequently reaches between 13 and 18 per cent., but in not a few epidemics it is as high as 25 per cent., or may even reach between 30 and 40 per cent.

Waidele found that the mortality of his patients was 12 per cent.; Köstlin, of Stuttgart, lost in 1853, 12.9 per cent.; Senfft, in 1862–3, 13⅓ per cent.; Reisinger lost, of 120, 16, or 13⅓ per cent.; Russegger, of Kitzbüchel, in Tyrol, in 1847–8, lost 14 per cent., so also did a physician in Remagen; Höring, in Heilbronn, Krauss, in Walddorf (31 out of 226), and Rösch, in Schwenningen (39 out of 261) lost 15 per cent.; Faber, of Schorndorf, in 1825–7, lost 73 out of 460, or 15.7 per cent.; Bidder, in 1831, in Courland, lost 15.6 per cent.; Voit, in 1867, in Würzburg, lost 16 per cent.; Wünstedt, in 1864, in Copenhagen, lost 16 per cent.; Barthez, 16.6 per cent.; Huber, in Memmingen, in 1869, lost 17 per cent.; Gläser, in Hamburg, lost 23 out of 118, or 19.4 per cent.; Gauster, in 1856–7, in Stein, lost about 20 per cent.; Feitel had a mortality of 23.3 per cent.; Böhmen, according to Löschner, lost, in three settlements, 445 out of 2,057 patients, or 21.8 per cent.; Stuttgart, in 1862, according to Köstlin, lost 23.8 per cent.; Hof, according to Marc, lost, in 1842, 22⅓ per cent.; in 1862–3, of 450 attacked, 127 died, or 28 per cent.; Greifswald, in 1850–1, according to C. Mosler, and in 1860, according to Ziemssen, lost about 25 per cent. The highest figures are the following: Hambursin, in Namur, lost about 30 per cent.; Arrigoni, about 40 per cent.; Salzmann, in Esslingen, between 1853–7, about 36 per cent.; at Hornbach, in the Palatinate, in 1868–9, 34 per cent. died. Compare in this respect especially the statistical report of Majer.

Comparatively benign and malignant epidemics often alternate with one another in the most varying manner, in one and the same locality, without assignable cause; indeed it often appears, as if the changing character of the disease, which is now mild and now severe, did not only vary from epidemic to epidemic, but with many decennia, or with periods embracing whole lifetimes.

The fluctuations in the severity of the epidemics do not occur at the same time in different places. According to Köstlin the following astonishing facts have been observed in Stuttgart: after 1830 scarlet fever disappeared entirely, and the first epidemic which at length appeared was in 1846, and there were no fatal cases. Then in the following epidemics of 1853, 1856, 1862, and 1867–8 the mortality in the entire city was respectively 72, 149. 182, and 14; the physicians of the poor had a mortality of 12.9 per cent., 7.3 per cent., 23.8 per cent., and 5.2 per cent., so that there were variations in the mortality between zero and 23.8 per cent., and besides this there was an interval of fifteen years in which no cases occurred. Förster says that in Dresden, for the fifteen years between 1794 and 1808, and throwing the epidemics out of the account, one out of 29 deaths among children was from scarlet fever, and so between 1823 and 1837 there was one in 28; on the other hand, in 1862, during an epidemic of scarlet fever, but one died out of 86; so that a very apparent improvement had taken place.

It may be very questionable whether the season of the year has an influence on the character of the epidemics. Malignant epidemics of short duration have appeared at every time of the year, and when such an one has lasted for a considerable period of time, it has generally been quite as severe in the summer as in the fall and winter.

According to Ranke, the fatal cases of scarlet fever in Munich, from 1859 to 1868, were scattered over the different seasons of the year in almost precisely the same manner as the disease itself, which shows that the season does not exert a special influence over the course of the disease.

Tripe, reckoning from extensive material (11,566 fatal cases in London between 1848 and 1852, with 1,575 fatal cases from dropsy) instituted an inquiry as to whether death from dropsy was associated with certain seasons of the year. In the first quarter the number of such deaths was 268 out of 2,162, or 13 per cent.; in the second, 266 out of a total of 2,279, or 12.3 per cent.; in the third, 322 out of a total of 2,930, or 10.9 per cent.; in the fourth, 719 out of a total of 4,195, or 17 per cent. Calculating the percentage of deaths from scarlet fever and dropsy by the single months, the results were, that the mortality of the first five months, also of July and August, fell below the average, while in June and the last four months it was above it. Judging from these statistics he believes that at certain times there is a special activity of certain factors which augments the intensity of the poison, or affects the kidneys more severely, or makes dropsy more fatal. As may readily be seen, the differences in the ratios are not very great.

The question as to which show the greater mortality, the epidemics of the city or those of the country, has been answered differently by different writers. Richardson found it was greater in the cities than in the country, while Eulenberg, and especially Kolb, found it was decidedly less. In settling this question, there are certainly other factors than those commonly recognized, such as the density of the population and the availability of medical aid. The latter may be better, and accomplish better results in the cities than in the country, while, on the other hand, the country is more exempt from the evils of overcrowding. Meanwhile, when we take into consideration that some localities are deeply infected, while others in the neighborhood, under the same conditions as regards population, remain free or suffer but little, the more probable view is, that the medical art will more than make up for the differences mentioned, and it is quite likely that the differences that have been observed between city and country are somewhat accidental, and dependent chiefly on the situation of the dwelling.

The influences of *poverty* and *opulence* upon the fatality of scarlet fever are not so cogent as have been supposed. Of course the wretched homes of the poor, and the entire absence of nursing and care, cause an increase of danger which those in better circumstances are able to avoid; but of far more importance as a prognostic indication is the degree of personal predisposition to scarlet fever, or, in other words, the degree of resistance to the influence of the contagion, which is not materially affected by external circumstances. Very frequently cases which are severe at first terminate fatally, notwithstanding the best of care and treatment. The beneficial effects of favorable surroundings are seen, not so much in their therapeutic influence during the course of scarlatina, as in their prophylactic power to prevent the spread of the disease.

As a matter of course, *good nursing* and *proper medical advice* will lessen the percentage of mortality. It is probable that in many of the epidemics of earlier times, a greater mortality was due to the erroneous therapeutical notions then in vogue than would have been caused by the average severity of the cases if left to themselves. The excellent effect of good nursing is most strikingly realized in the observations of Shingleton Smith, who, while at the same time attending a district and a hospital, observed, that not only in respect to the mortality, but also as regards the sequelæ and complications, the results were far more unfavorable among the cases treated at their homes than among the cases which were removed to the hospital and received proper care and nursing. Of course, if a hospital is unfavorably situated and improperly administered, the beneficial influence, which it would by proper care and treatment certainly exert upon the cases, will be more or less diminished.

Sex has no effect upon the mortality of scarlet fever. If in some places the mortality is greater among males, in others it is reported as greater among females. Striking differences of this character are probably due to local variations in the number of male and female persons of the age most subject to scarlet fever.

Richardson collected from the decade 1838–1848, 102,382 fatal cases of scarlet fever, of which 51,660 were males, and 50,722 were females, a very slight difference in such a large number, and moreover, fully accounted for by the surplus of the male population in those districts in which the cases occurred. Passow observed that of 1,579 fatal cases of scarlet fever in Berlin, in 1863 and 1867, 795 (50.35 per cent.) were males, and 784 (49.65 per cent.) females; the difference being only 0.02 in favor of the males.

Of all the conditions which increase the danger and mortality of scarlet fever, *age* is by far the most potent. Notwithstanding the opinion of Faber, that nurslings are lightly affected by the disease, most other authorities consider that among them the severer forms of scarlet fever are more frequently met with than among older children. The smaller number of fatal cases during infancy is solely due to the slighter predisposition of infants, in consequence of which they frequently escape the contagion. The following years, up to the fifth or sixth year, are the most dangerous, and have a pretty equal mortality. With the increasing years of childhood the prognosis of scarlet fever becomes decidedly better, though not by any means in the same measure as in measles. Among the few adult cases the mortality is greatest among pregnant and puerperal women and invalids.

Fleischmann estimated the mortality of the first year (8 cases with 6 deaths) at 75 per cent., of the second to the fourth year (204 cases, 88 deaths) at 43 per cent., of the fifth to the twelfth year (260: 51) at 19.6 per cent. Wasserfuhr did not observe any decrease of mortality from the second to the sixth year. Russegger found the mortality up to the fifth year (101 : 21) = 20 per cent., from the fifth to the tenth year (126 : 20) = 15 per cent., from the tenth to the fifteenth year (47 : 3) 6 per cent., over fifteen years (27 cases) no one died. Waidele found the highest mortality up to the second year, and the eleventh year was very dangerous. The following rates of mortality were observed by Krauss in the epidemic which occurred in 1854 in the villages of Walddorf and Oferdingen: in the first year (13 : 4) = 30 per cent., in the second to the third year (33 : 9) = 27 per cent., in the fourth to the sixth year (80 : 21) = 26 per cent., in the seventh to the ninth year (62 : 7) = 11 per cent., in the tenth to the twelfth year (44 : 3) = 7 per cent., in the thirteenth to the twentieth year (40 : 2) = 5 per cent. The 280 fatal cases reported by Voit are distributed thus: first year (5 : 1) = 20 per cent., second to the sixth year (166 : 24) = 14.4 per cent., seventh to the sixteenth year (109 : 10) = 9.1 per cent. In the experience of Ranke the aggregate mortality of scarlet fever was 6.8 per cent., while that of children under one year of age was 10.7 per cent. In Zehnder's experience 66 per cent. died in the first year, while in the following years the rate of mortality sank rapidly to 31, 23, 12, 10 per cent., rose in the sixth year to 20 per cent., and in the eighth again (from 9 per cent. in the seventh year) to 17 per cent., and from the tenth year onward, among 64 cases, only one, a girl of twelve years, died. According to Rösch, out of 17 cases under one year of age, 5 died, out of 156 cases of from two to five years of age 31 died, and of 88 cases over five years, only 3 died.

According to Tripe, among the fatal cases of scarlet fever those due to dropsy are epecially frequent among children of three to five years of age, less so among older children, and least of all among those under one year of age.

The influence of the mortality of scarlet fever upon the general mortality varies materially with the frequency of the disease, and especially with its degree of malignancy in different localities.

Thus, according to Schiefferdecker, in Königsberg, during years of severe epidemic, as 1860 and 1867, there were in 1,000 deaths 91.12 and 119.60 due to scarlet fever; while in years that were free from epidemics, as 1858, 1866, and 1862, only 0.94, 4.76, and 6.0 were owing to that disease. In Berlin, according to the same authority, between 1843 and 1860, the years of severe scarlet-fever epidemics, 1852, 1854, and 1856, had in 1,000 deaths 38.44, 23.90, and 33.51 due to scarlet fever; while years which were free from epidemics, such as 1858, 1859, and 1860, only 2.20, 1.96, and 4.31 were due to this disease. In Frankfort-on-the-Main these figures varied during the years 1853–1864, from 0 (1864), 0.90 (1855), 1.61 (1860) to, on the other hand, 103.20 (1862); in Munich, 1862 to 1868, from 1.0 (1865) to 14.58 (1868); in Stuttgart (1853 to 1867) from 0 (1861), 0.93–2.28 (1858 to 1860) to 100.76 (1862) and 109.27 (1856); in London (1861 to 1866) from 23.51 (1866) to 69.73 (1863).

If the averages for the reported years in the different cities be compared, we find that in 1,000 deaths in London (during six years) scarlet fever caused 42.10; in Königsberg (12 years) 31.70; in Stuttgart (15 years) 23.73; in Frankfort (13 years) 17.29; in Berlin (18 years) 12.92; in Munich (7 years) 6.34; in the canton of Geneva (13 years) 4.92.

Schiefferdecker, moreover, collected the maxima of these individual cities and found as follows: Königsberg, 1867 (119.60 deaths from scarlet fever in 1,000 deaths); Stuttgart, 1856 (109.27); Frankfort, 1862 (103.20); London, 1863 (69.73); Berlin, 1852 (38.44); Munich, 1868 (14.38). Hence we learn, provided the figures of the years of epidemics were not at the same time materially influenced by an unusually large aggregate of deaths from other diseases, that, among the cities named, Königsberg, in 1867, had the severest epidemic of scarlet fever; Stuttgart and Frankfort were visited only rarely, but then by very malignant epidemics, whilst London suffered from the disease uninterruptedly, but never from any serious epidemic. These facts agree with the general law, that large cities suffer less from epidemic diseases than small ones (Berlin and Munich likewise have low averages).

The disproportionately large mortality among children is shown by the following statistics:

For the above-named cities Schiefferdecker found that in 1,000 deaths the proportion of persons under fifteen years of age dying of scarlatina was, in London, 80.80; in Königsberg, 63.44; in Frankfort, 51.37; in Stuttgart, 46.35; in Berlin, 24.77; while the deaths from the same disease among persons above fifteen years numbered, in London, only 3.67; in Königsberg, 1.22; in Frankfort, 1.06; in Stuttgart, 1.56; in Berlin, 0.77.

The following table exhibits the mortality from scarlatina per 1,000 total deaths, and is arranged by single years and by periods of childhood:

Years.	Königsberg. 1863–68.	Berlin. 1843–60.	London. 1861–66.	Frankfort. 1862.
0–1	5.76	2.50	10.89	3.88
1–2	50.80	16.00	58.34	(1–5)
2–3	135.20	45.78	137.41	88.81
3–4	176.00	90.33	212.31	....
4–5	231.60	102.50	248.52	....
0–5	45.00	15.64	64.53	33.00
5–10	223.08	120.13	256.57	216.08
10–15	155.02	40.90	109.90	118.22
15–	1.22	0.77	3.67	1.06

TREATMENT.

The prophylaxis of scarlet fever can only be accomplished by the prompt removal of the healthy from the sick, or where this is impossible, by the seclusion of the latter, and the destruction, or at least complete and satisfactory disinfection, of every article that has come in contact with them. Hence the patient must have a separate room, to which only his attendants should be admitted. All useless articles of furniture should be removed from the patient's room, such as musical instruments, bookcases, and cupboards, everything which can collect and retain dust and dirt (curtains, above all woollen ones, carpets, etc.), and all small articles. Provision should be made in some way for ventilation of the apartment by means of an open window, or, should the weather not permit of this, by one in an adjoining room, the door between remaining open. The patient should be properly protected from any strong draught, which, however, need not be particularly feared. Further, the patient should be kept perfectly clean, and every article used by him should be thoroughly disinfected: the expectoration, urine, and excrements should be received in vessels containing disinfecting fluids (chlorinated lime, carbolic acid), and frequently removed. The moment the patient's linen is changed it should be placed in a similar fluid, and then thoroughly washed in tubs used only for this purpose; articles of slight value should be burnt, for instance, the pieces of linen which Budd recommends in lieu of handkerchiefs; the sweepings of the apartment should also be destroyed by fire. In like manner it is necessary to have the articles used by the attendants, and especially their clothes, washed or disinfected in the same way as those of the patient. The best mode of disinfection for articles which cannot be washed is to expose them to a high degree of heat, and then give them a thorough airing. The nurses should be ordered to wash their hands frequently in some disinfecting fluid; they should keep their persons thoroughly clean, and change their clothing whenever they have to come in contact with a third person, and especially when they enter the service of another patient. To prevent the dissemination of the dusty particles of the infecting epidermis scales, particularly during the period of desquamation, it is of the utmost importance to keep the whole body perfectly clean. Budd recommends that the body, including the head, should be rubbed twice daily with olive oil, especially when bathing is impossible. The convalescent should not be allowed to mingle with the family until desquamation has ended, and then not until his body has been most carefully washed and clothed in a fresh, clean suit; his apartment and all that has remained in it during his sickness, and everything that has been used, should be most thoroughly cleansed and disinfected. The walls should be repapered or painted, and before reoccupation of the room, the windows and doors should be allowed to remain open for a long time.

We have already, when speaking of the modes in which the contagion spreads, and in our observations concerning its persistency, shown the necessity for such rigorous measures, more especially when diphtheria has complicated the disease. In the accomplishment of complete isolation of the patient we can expect a much better result than in measles, owing to the slight degree of volatility possessed by the scarlet-fever poison. Whether the poison of scarlet fever can be propagated by means of sewer gases, is a question not yet settled. Druitt has urged the necessity of a proper construction of water-closets and sewers. Fergus has investigated this point, but his results are unsatisfactory, yet there can be no doubt that absolute cleanliness is very desirable.

A complete isolation of those localities and districts suffering from scarlet fever is, owing to the commercial relations of the present day, impracticable, or is only possible under the most favorable circumstances, as in the case of an island, etc. It is even questionable whether the closing of houses containing cases of scarlet fever, as has been proposed by Reil, could be carried out; at any rate, this measure is impracticable in cities. In Christiania it has been proposed to post a notice upon the infected dwelling.

All patients who cannot be properly treated at home should be removed to hospitals set apart for contagious diseases, the scarlet-fever patients being placed of course in separate wards, where suitable attention can be secured. In order to avoid the spreading of the poison, wagons or stretchers devoted exclusively to this purpose should be used for transporting such patients. Upon the outbreak of scarlet-fever epidemics in the country, inexpensive barracks may be constructed, such as were used by Mosler in a typhus-fever epidemic. Furthermore, in an appropriate climate camps for the sick, if favorably situated, are advisable.

It is, moreover, desirable that the patients should be separated from each other, or that at least many should not be together in the same room, for experience has repeatedly shown that the neglect of this precaution has increased the severity of the individual cases. Thorough ventilation is consequently of the utmost importance.

To prevent the spreading of scarlet fever by means of well persons, it is perfectly justifiable to deny to the brothers and sisters of the patients entrance to nurseries, institutions, and schools, and this prohibition should remain in force until the complete disappearance of every symptom of the affection of the throat and skin. The State should be responsible for the thorough execution of these all-important measures during a scarlet-fever epidemic. They are of far greater moment than in the case of measles, not only because scarlet fever is more dangerous than the latter, but also because with the increasing years of childhood the predisposition to scarlet fever very materially lessens, and hence it is possible that children may entirely escape the disease if they be secured from exposure during early life. Therefore, the compulsory notification of every case of scarlet fever is of the utmost importance. On account of the minority of those really susceptible to the disease, it is generally unnecessary to close the schools upon the breaking out of an epidemic of scarlet fever, and such a measure should be insisted upon in isolated communities and districts only when the epidemic happens to be unusually extensive and malignant.

Finally, all display should be prohibited at the funerals of those who have died of scarlet fever, and even upon any other occasions, so long as scarlatina continues in the house, especially if the epidemic be of a severe and virulent type. Children should not be allowed to take part in these funeral ceremonies, and the opening of the coffin in the presence of the assembled friends, as is customary in many places, should be strictly prohibited.

To the isolation of the patient, when the epidemic is mild, or the case an idiopathic one, the following objections have been made. In the first place, the process is very inconvenient, and often fails of success if it be incompletely carried out. If it be omitted, the other children will quite likely take the disease in the same mild form, and thus be protected against a later attack, which might be of a malignant character. Again, it is urged that it is better to have scarlatina in early childhood, rather than in later years when the patient may at great loss be suddenly and for a long time taken from his business, and when the danger from the disease is fully as great. Finally, even the most rigid isolation and most careful disinfection will not prevent the retention of the poison in the house; the other children who have been isolated for perhaps weeks may contract the disease upon the renewal of the exposure, and the quiet of the household has again to be disturbed; while if isolation were not adopted all the children liable to contract the disease would go through their illness at about the same time, and the care of them would therefore be less onerous. Notwithstanding these objections, no physician should advise or approve of the sick remaining among the healthy. Even during an epidemic of a mild type, or when one or more members of a family have a mild form of scarlet fever, a well child may take the disease in its malignant form and die, or may survive with some irremediable sequel, such as deafness. At any rate, should the first case prove of a malignant type, isolation should certainly be enforced, and all delicate children should be removed from the influence of the poison of scarlet fever.

There are no other prophylactic measures except isolation and the disinfection of everything contaminated by the contagion. The good result of any kind of inoculation is exceedingly doubtful, and still less worthy of recommendation is that old, much-vaunted pseudo-prophylactic, belladonna, or any other similar narcotic, while the internal administration of carbolic acid, lately recommended (Giersing) is as yet untried. Mouth washes and gargles are likewise of very dubious efficacy; such, for instance, as very strong dilutions of muriatic acid, which Godelle proposed as a means of destroying the poison of scarlet fever at the point where it is supposed to enter the body.

A common mode of treating scarlatina is by the internal administration of substances which are supposed to destroy the poison as soon as it has manifested itself in the system, such as acids, organic (acetic) and mineral (muriatic, sulphuric), chlorine, and especially the carbonate of ammonia, combined with the external use of the same means (vinegar and chlorine lotions). While very valuable and reliable antimiasmatic properties have been ascribed to these measures by individuals, the experience of many physicians, in large numbers of cases, has not been able to confirm them. Perhaps the warm praises bestowed upon them may be explained by their having been used only during mild epidemics, when nearly all the cases get well of themselves.

The efficacy of the non-specific methods of treatment, so highly advocated by many, is entitled to no greater confidence. They have perhaps in one way or another proved beneficial in individual cases or epidemics, but no one of them has succeeded in obtaining general approval.

The following are those most worthy of mention:

Bloodletting, general as well as local, and carried out with more or less energy, has been looked upon by many physicians as the most certain means for reducing the mortality to a minimum. Extensive experience, however, has shown it to be a dangerous remedy, only to be resorted to under certain circumstances for the removal of individual casual complications. In malignant cases, more or less energetic bleeding has not only not produced any decided beneficial result, but has hastened the threatening collapse. The mild cases of the disease will recover without, and even in spite of, blood-letting.

The indiscriminate administration in large quantities of *laxatives* and *emetics*, in accordance with a theoretically conceived method, is highly objectionable. It is impossible for one to know beforehand whether the case may not present such marked intestinal symptoms that the use of powerful remedies of this character would prove very detrimental. Furthermore, as many a patient has died notwithstanding powerful purging, whether spontaneous or the result of remedies, this method of treatment seems entirely worthless.

Anointing the body with lard was first recommended by Dähne, and later by Schneemann. The latter, in cases of scarlet fever, has the whole body except the face anointed twice a day for three weeks, and once a day during the fourth week. Besides this, he orders the sick-room to be kept at a low temperature (61° Fahr. at the most), free ventilation, no medicines; the patient to avoid going to bed and to move about as much as possible, and after the tenth day to go out of doors regardless of the weather. He declares that with the practice of his method no regular desquamation occurs, but merely the ordinary natural shedding of the epidermis. Hence, when the redness of the skin has disappeared the disease is supposed to have run its course, notwithstanding the fact that the period of desquamation is the most dangerous not only on account of the contagiousness of the disease but also by reason of the complications and sequelæ. This method of treatment has never been strictly followed out to any extent, although some of its individual features have been adopted more or less completely by many physicians; but in malignant cases experience has proved that the treatment by the anointment method is as ineffectual as any other.

The old-fashioned general treatment for all the exanthematous diseases, which sought by any means to produce diaphoresis, is simply preposterous, and although regarded as detrimental by physicians, and therefore entirely abandoned by them, has unfortunately remained in such general use among the people that it is not unfrequently a great obstacle to the proper practice of the more rational therapeutics of the present time.

The *symptomatic* is the only rational and advisable treatment for scarlet fever. In its application it must constantly be borne in mind that the disease, which cannot be cut short or interrupted in its natural course, always tends to recovery, provided the fever and local symptoms remain within certain bounds. Hence every feature of the disease should be most carefully watched, in order that with the appearance of any anomaly the necessary interference may be resorted to.

During the normal and uncomplicated course of scarlet fever the following plan of treatment is to be followed. The patient should remain in bed from the beginning of the disease until the completion of desquamation. The bed coverings should not be oppressively heavy nor of too great warmth. The situation of the bed should be such that, without compromising the thorough and constant ventilation of the room, the patient will not be exposed to any strong draught, which might possibly be injurious to him. The temperature of the room should be a low one —59° Fahr. is amply sufficient; a higher one is only necessary when the over-sensitive patient really complains of feeling chilly, which is only likely to be the case when, with the decrease of the fever, a cooler temperature is no longer needed to counterbalance the effects of an excessive production of heat.

In scarlet fever it is particularly necessary to pay some attention to the skin; for instance, baths should be used in the manner described farther on, or, if they are impracticable under the circumstances, other hydrotherapeutic measures may be resorted to. At all events the body should be sponged two or three times daily with cold or tepid water, which at the same time will be found very effective in soothing or removing the annoying itching and burning of the congested skin. The linen of the patient should be frequently changed. During the angina it is sufficient for the patient to use a slightly astringent gargle, and to rinse his mouth with some cleansing fluid. Let him have fresh water or lemonade to drink. During the height of the disease the diet should be restricted, without excluding, however, such light alimentary articles as milk and thin soups, especially in the case of young children, who perhaps have some desire for food; during the decline of the disease good nourishing food may be allowed, although overloading of the stomach is of course to be avoided. During the whole affection care should be taken to keep the bowels regular, either by the judicious administration of fresh or stewed fruit, or possibly by the use of a mild aperient, calomel excepted. The physician should not allow the patient to leave his bed until the process of desquamation is complete, or at any rate not before the end of the third week, lest by permitting him to get up too soon he incur the blame of having caused the nephritis which may come on later. He should also examine the urine daily, both microscopically and chemically, in order to recognize the kidney affection early, and so to obviate, as far as possible, its dangers.

A very serious question nowadays in the treatment of scarlet fever is, whether or not under all circumstances a hydrotherapeutic course shall be pursued. A very high degree of fever, occurring in the course of an otherwise perfectly normal case, of course necessitates the use of cold water as much as do the high fevers of other diseases (measles for instance, page 120, et seq.), partly to afford the patient personal relief, and partly to ward off those real dangers which an excess of fever entails. Yet in cases which run a mild course, with only a moderate amount of fever, the antifebrile hydrotherapeutic measures may seem unnecessary. But it has been stated that the cold-water treatment, if begun early in the disease, and continued regardless of the intensity of the fever, is able not only to entirely cure the disease, but also to protect against the localization of the scarlet-fever poison in the kidneys. If these facts were true, they would certainly show a most brilliant and important therapeutical result; but unfortunately the assertion in question seems to have no trustworthy foundation. The various complications and anomalies in the course of scarlet fever can by no means be prevented by hydrotherapeutics, for cases have been reported in which, notwithstanding a perfectly appropriate water-treatment during the febrile exanthematous stage, an extensive dropsy set in, and finally carried off the patient. The results also of my own experience—chiefly, it is true, in private practice, in which the thorough execution of the cold-water treatment is unfortunately not so practicable as in a well-equipped hospital—do not corroborate the truth of the above-mentioned statement. It seems therefore advisable, at least for the present, to practice hydrotherapy energetically only in the severe cases; in the meanwhile trustworthy statistics should be collected from various epidemics and districts (for the frequency of nephritis varies in the most extraordinary manner), upon which a definite judgment may be based concerning the treatment of the lighter forms of the disease.

In any case it is judicious to bathe scarlet-fever patients; the d prejudice, so prevalent against baths, and based upon the ar lest the patient may thereby take cold, must be laid aside. feel convinced that the nephritis has been less frequent, and of decidedly lighter character, since I have ordered a daily bath, han before I was in the habit of prescribing it. This would lead o the conclusion, that if early in the disease the skin receives roper care the congestion of the kidneys, which is of such fre- uent occurrence, may perhaps be prevented altogether, or cer- ainly lessened, and thereby dangerous consequences avoided. t all events, the temperature of the baths should be graduated ccording to the intensity of the fever. During the period of igh fever, in the beginning of scarlet fever, Ziemssen's baths, n which the water is gradually cooled down, should be used; nd in the following days, as the fever sinks, the temperature f the baths should gradually be increased; during convales- ence the baths should be warm. In cases of mild scarlet fever, hen the children are especially sensitive to cold, and the arents very apprehensive, it is best to use only the warm baths, earing in mind, however, that such considerations should have o influence in preventing the carrying out of the appropriate nd correctly administered cold-water treatment, which alone is uitable in serious cases with an intense degree of fever. Bagin- ky advises that after the tepid bath the cases with very high ever should be quickly and thoroughly dried, and then anointed ith lard and allowed to lie down with only a light covering; he urther recommends that during the period of defervescence a light diaphoresis be kept up for about two hours, between the arm bath and the anointing with lard, during which interval, f course, the patient must be guarded against taking cold hrough any unnecessary exposure.

Although in simple and therefore typical cases of scarlet ever the above-described dietetic and expectant mode of treat- nent is sufficient for the abatement of present and the prophy- axis of further disturbances, yet there are *anomalous* and *com- olicated* cases which require not only a strict carrying out of the bove-mentioned measures, but also the employment of further neans.

An intense degree of fever is generally the most frequent indication for some energetic interference, and is most appropriately met by the combination of active hydrotherapeutic measures, with antifebrile medicaments, and in individual cases these may be supplemented by anointing the body with lard or with carbolic acid salve (Betz), a plan which is of special benefit when the skin is very dry and hard. Literature is rich in the records of cases of scarlet fever with a high degree of fever, accompanied by serious disturbances of the nervous system, which have been successfully treated by means of pretty cool baths, properly administered packs, the cold douche, or the more or less general use of cold dressings; in like cases, therefore, no rational physician should hesitate to employ hydrotherapeutic measures. Among the medicaments quinine is of special value, in doses of eight grains (in adults as high as fifteen grains), which may be given, except in the case of very young children, two or three times daily, during one or two days; these large doses, however, must not be given for several days in succession, on account of the toxic effects of the drug. Digitalis, in doses of from seven to thirty grains daily, according to age, is of service in reducing an extreme frequency of the pulse.

Energetic measures for a reduction of temperature are not only necessary when the fever is for a longer or shorter period of an intense nature, but they should also be employed in those cases in which the fever, whether of a high or low degree, is continuous and disproportionately prolonged, without the existence of any local trouble which might explain its persistence. The appropriate treatment in these cases should be that above described, although the doses should be smaller and the hydrotherapeutic measures less frequently repeated.

Whenever a highly febrile condition assumes a constant character, there should be no hesitation in anticipating a state of extreme exhaustion, which may set in suddenly, by the timely and frequent administration of nutriment, although an utter lack of appetite and great embarrassment in swallowing may render the accomplishment of this very difficult. For this purpose Hare recommends frozen beef-tea, in order at the same time to fulfil an indication for the pharyngitis.

An endeavor should be made to meet those symptoms due to the toxic effects of the scarlet-fever poison by the administration of antiparasitic and antizymotic remedies, while symptoms of collapse may be met by powerful stimulants. Among the former I will mention quinine, in medium and large doses, the internal and subcutaneous use of carbolic acid, the sulphocarbolate of soda (in daily doses of from fifteen grains to a drachm, according to the age, as recommended by Sansom), the hyposulphites, and the inhalation of ozone (Francis); to the latter belong especially camphor, benzoin, musk, and the carbonate of ammonia, as also rum, Cognac, and the stronger wines; and finally, cold affusion in the empty tub or while in the bath. In desperate cases, moreover, it may be worth while to practise transfusion of blood. Bennett reports that by the administration of fresh yeast, from one to two tablespoonfuls, repeated several times during the day, according to the age of the patient and the malignancy of the disease, he has never lost a case of malignant scarlet fever.

The symptoms appertaining to the skin, as a rule, no longer require such active treatment as in former times, when the greatest importance was attached to the eruption. It will, for the most part, suffice to fulfil the general indications, should the eruption show any irregularities; it has, moreover, been proved that by the application of the hydrotherapeutic measures the fluxion to the skin is in no way interfered with, but frequently rather favored. Thus the eruption has frequently been observed to come out, or to increase, when, on account of a high degree of fever, the patient has taken a tepid bath with cold affusions, or has been in a pack. When the eruption is slow in developing, it may frequently be hastened by the use of mustard poultices, and by anointing the skin with lard; and if the fever be moderate, diaphoretics may also be tried. The same means are frequently of use in prolonging for a time an eruption which has a tendency to fade too soon; and such a measure should certainly be attempted if dangerous symptoms appear imminent. If the latter really come on, and these simple means do not suffice, there should be no hesitation in employing more powerful ones. For this purpose may be employed, externally, the warm bath as recommended by Wunderlich, warm and hot douches, simple hot poultices containing some mustard, the cold pack, and cold affusions, followed by the use of warm wrappings; and internally, the diaphoretic infusions, ammonia and musk. Max Langenbeck speaks in the highest terms of the use of the hot flat-iron, combined with a mustard bath, followed by a sweat brought about by warm wrappings.

Severe brain symptoms are generally only due to the fever caused by the specific poisoning, and will yield to active antifebrile treatment in proportion as the latter is able to control the fever. Congestion of the brain, incipient meningitis, etc., when diagnosticated, demand energetic antiphlogistic measures; the whole head to be covered with a bag of ice, the application of cold dressings, leeches to the forehead and behind the ears, and possibly venesection. Unfortunately brain trouble cannot always be recognized with a sufficient degree of certainty to render advisable a more energetic blood-letting, which might prove beneficial. Narcotics, in doses suitable to the age, are appropriate in cases of simple brain excitement where there is no suspicion of hyperæmia, and where no improvement has followed the use of cold derivatives, etc.; and they are of great value in nervous delirium, during and after the period of defervescence.

As long as the angina remains at all moderate, and gives rise to no dangerous symptoms, it is sufficient to do nothing special beyond the frequent application to the neck of cold dressings, or those recommended by Priessnitz, the taking of pieces of ice into the mouth, and the use of detergent mouth washes and gargles; for very often, in the case of young scarlet-fever patients, any further local treatment is impossible, or at least throws them into the greatest excitement. The application of leeches to the neck, or cauterization of the tonsils with nitrate of silver, in strong solution (thirty grains to the ounce), or in substance, is only necessary when, in the cases of children, these glands rapidly enlarge and threaten suffocation; while in older patients pretty free scarification of the tonsils may also be practised. Gangrenous affections of the throat are to be treated with disinfecting and deodorizing gargles (permanganate of potash, fifteen grains to the ounce of water; chlorate of potash, chlorine water, carbolic acid, one part to two hundred of water), and with cauterization of the ulcerated surfaces. Complicating diphtheria should be treated like simple diphtheria, either in a simple manner or with weak or concentrated disinfectants, etc. (compare p. 128). If the diphtheria extend into the larynx, tracheotomy can afford but little hope, for I have never seen the operation succeed during scarlet fever. In diphtheria of the nasal fossæ, an effort should be made to remove the membrane mechanically, or by means of solvent and softening agents (lime-water), as well as astringent and cleansing injections, especially if there are any ulcerations.

A similar plan of treatment should be pursued in the case of simple coryza or purulent ulcerative coryza. In young children the nares should always be oiled or greased.

Any kind of stomatitis demands, besides thorough cleansing of the oral cavity and timely cauterization of the ulcerations, an absolute disuse of any form of mercury, on account of the danger of causing noma.

The treatment of disease of the ear should never be postponed until convalescence has set in. Wendt advises that from the beginning the secretion should be very carefully removed from the nose, throat, and the lower portion of the Eustachian tube by means of douches and gargles, and from the external auditory canal (after spontaneous or artificial opening of the membrana tympani) by means of injections. The forcing of air into the middle ear is a measure of very great importance (of course, not by means of the catheter). The frequent repetition of these procedures will, when the swelling has nearly, if not quite, obliterated the canal, guard against the formation of adhesions between the two mucous surfaces. At first, for the pain, leeches should be applied, while later, poulticing is more effective (Wendt). Disease of the ear does not contraindicate cold baths, but during their use the auditory canal should be closed with oiled cotton.

For severe conjunctivitis, the assiduous application of cold-water dressings is necessary. In severe cases of keratitis the spontaneous rupture of the cornea may frequently be prevented by the local application of intense cold, the use of atropine, or finally by puncture of the cornea. In such cases it is proper to keep the eye closed; some endeavor should be made to remove the secretion, but with struggling children no force should be used, lest the dreaded perforation be thereby produced. As a result of purulent keratitis the extirpation of the globe of the eye may finally become necessary (Senfft).

Intense inflammation of the cervical cellular tissue, with or without severe lymphadenitis, demands the application of ice or cold-water dressings, while leeches should only be used in case of necessity. As soon as the formation of pus and fluctuation are demonstrable, the cold-water dressings, as far as the general condition will allow it, should be changed for warm ones, and the abscess speedily opened, in order to guard against dangerous burrowing, erosion of large vessels with fatal hemorrhages, or the destruction of important nerves. During the treatment of the abscess, as well as during its evacuation, disinfectants should be made use of. Glands that are the seat of chronic infiltration require the external use of absorbents, iodine, etc., as in the case of simple glandular affections. The moment disease of the cellular tissue shows signs of becoming gangrenous, some powerful caustic should be made use of (Hebra recommends caustic potassa or a concentrated acid or the nitrate of silver) and the wound assiduously dressed with preparations of carbolic acid. The secretions from such wounds and all gangrenous shreds or sloughs should be frequently removed. The internal administration of quinine and acids, as well as stimulants and strong nourishment, with wine, is most urgently indicated.

Every case developing diphtheria or gangrene should be separated *at once* from the other scarlet-fever patients. For that very rare occurrence, hæmatoma of the neck, the indications are, according to Huber, the application of ice and ligation of the carotid.

The presence of bronchial and pulmonary complications renders a pure and constantly renewed air of the utmost importance. Aside from its other advantages, such an atmosphere strengthens the respiratory act, and thereby the expectoration of the secretion is very materially facilitated and the dyspnœa relieved. Otherwise, with the avoidance as far as possible of the abstraction of blood in any form, the treatment is the usual one;

stimulation of the heart's action, maintenance of the strength, antifebrile measures, and the judicious use of expectorants. In cases of œdema glottidis or stoppage of the air-passages from other causes, diphtheria excepted, tracheotomy is, not unfrequently, perfectly successful. Warm baths, and if necessary the application of hydrotherapeutic measures (compare p. 124), are often of material aid in determining a favorable issue to the lung affection.

Intestinal disturbances, generally diarrhœa, do not as a rule require any internal remedies. They should be treated, especially when the fever is high, with frequently changed ice-water dressings or with an ice bladder over the abdomen, and the cold packs, described on page 125, are likewise of great benefit. A careful diet is of special importance, as in the intestinal affection of typhoid fever. If there is constipation, enemata should be given, and if necessary any mild purgative, excepting calomel, or in fact any of the mercurials. A moderate diarrhœa coexistent with dropsy is rather to be desired, and requires no active treatment.

Rheumatism of the joints is generally of a mild character and of short duration, and may require, besides an anæsthetic liniment and rest, some form of support like a snug bandage, but, as a rule, no further treatment. If it should result in a synovitis, as now and then occurs, with or without the formation of pus, the case should be treated in the ordinary way, beginning with the application of dry cold and the abstraction of blood, as in all severe joint inflammations.

Upon the first symptoms of inflammation of either of the serous membranes the thorough application of cold should be tried, to prevent an excessive exudation; the abstraction of blood is only allowable when the patient is robust—a condition rarely met with—and when the diagnosis is without doubt, which is not always the case in meningitis. Pleuritis with an excessive or purulent exudation should be relieved by paracentesis, performed by puncture or incision, under the same indications which authorize the operation in primary or non-infectious pleuritis; and this procedure, whichever way performed, has not unfrequently been followed by excellent results (Trousseau, Albu, Brotherston, Mühsam).

The treatment of endocarditis in scarlet-fever patients is the ordinary one; the continuous application of cold to the præcordial region, absolute rest, and digitalis for great frequency of the pulse.

Hemorrhagic scarlet fever requires the early employment of antifebrile and antimiasmatic means and methods; an abundant supply of fresh air and the administration of appropriate medicines, especially quinine; the hyposulphites, carbolic acid, and salicylic acid (five or ten grains to the ounce) are highly recommended. For the severe hemorrhages cold should be applied locally. Besides the internal remedies just mentioned, ergot, the sesquichloride of iron, and preparations of tannin have often been used with advantage. Compare, moreover, what has been already said concerning hemorrhages during measles (page 126). Wine, stimulants, and nourishment are all-important, and should always be comprised in the treatment.

After nephritis has set in, an aggravation of its intensity and the consequences may frequently be prevented by the continuance of the warm baths recommended for the period of defervescence, and by favoring a condition of diaphoresis; hemorrhages from the kidneys also will generally yield to these simple measures, so that there may be no necessity for resorting to any special remedy (hypodermic injections of ergot). Other far more active measures must, however, be promptly—for any delay would be dangerous—employed whenever a rapid decrease occurs in the amount of urine excreted (the latter containing an increased volume of albumen and blood), and dropsy has set in and is rapidly extending. In this case the results of experience indicate, as the most appropriate, an antiphlogistic treatment of the fundamental inflammatory affection of the kidneys. Hence, when dropsy sets in and rapidly increases, with a renewal of the fever, or an increase of that already present, accompanied by a full, slow pulse, and especially by pain in the region of the kidneys, with considerable hæmaturia (hydrops calidus), the treatment should be, for robust patients, one general bleeding (Romberg even bleeds in a child two years old; but copious bleeding may prove fatal!); for such as are less strong, a judicious local (in young children about three leeches, while adults had better be cupped) abstraction of blood in the region of the kidneys. If diarrhœa is not present, purgatives are indicated, in order, by their action upon the bowels, to relieve the kidneys; while diuretics, which increase the hyperæmia of these organs, and warm baths, which, if the fever is high, would increase it, should be avoided. If some improvement in the symptoms is not soon manifest, the abstraction of blood, but only locally, should be repeated; but in weak and anæmic children, derivatives in the region of the kidneys should be substituted (frequently repeated dry cups, and, less worthy of note, those agents which excite local inflammatory action, as blisters, and the like); the mild antifebrile and cooling hydrotherapeutic measures are also of service (cold packs, or moderately cool baths).

In many cases the abstraction of blood causes immediate and permanent relief; the fever and the pain in the region of the kidneys cease, the secretion of urine becomes augmented, the albuminuria lessens from day to day, and the moderate degree of dropsy that has been developed disappears. Now the warm baths should be renewed and a slight diaphoresis promoted, and a gradual but steady recovery will take place. In other cases, however, while the fever lessens or entirely ceases, the suppression of urine continues, the dropsy increases, and many other disturbances set in. In a similar manner dropsy of an extraordinary degree may finally develop in those cases which, during the beginning of the kidney affection, were attended with less fever and other alarming symptoms, but in which a high degree of albuminuria, and an abundant shedding of cylinder casts, with or without some hæmaturia, were present (hydrops frigidus). Here the thorough trial of warm, and in complete torpidity with excessive dropsy, even hot (that is, heated by the addition of water of 95° to 108° or 109° Fahr.) baths (Liebermeister, Ziemssen, Steffen), followed by about two hours of diaphoresis, will not unfrequently succeed in materially reducing the weight of the body. Like measures are indicated in anasarca, and when the peritoneal sac, the pleural cavities, or the pericardium are the seat of excessive exudations; and they are contraindicated in cases of capillary bronchitis and incipient œdema of the lungs, and also when eclamptic symptoms threaten and intercurrent attacks of fever occur, as under such circumstances sudden paralysis of the heart or dangerous convulsions may cause death. When the patient is very weak the water bath may be replaced by vapor baths in bed, or by hot-air baths as recommended by Bashan. Diaphoresis may be considerably aided by mild diuretics (juniper, ononis, acetate, tartrate or citrate of potash, lemon-juice, iodide of potassium, etc.), and alkaline salts, since, by their use, combined with the liberal administration of water, the secretion of urine is promoted, and the absorption of the effusions hastened. Whenever, notwithstanding an inconsiderable albuminuria, and a pretty copious secretion of urine, these measures are inappropriate or unsuccessful, either in very young children or in cases in which insufficient food and great poverty have brought about a condition of extreme anæmia, or where there is a profuse or long-continued intestinal catarrh, etc., a disappearance of the dropsy is frequently brought about by large doses of some good preparation of iron, which is also, for other reasons, indicated during convalescence.

Any excessive accumulation of fluid in either of the serous cavities may require the operation of puncture; this is considered by many (for instance Oppolzer, in case of ascites) to be accompanied by some danger, since during scarlatinous nephritis an inflammatory condition of the wound is more likely to occur. In a high degree of anasarca, scarifications should be practised, and the wounds covered by dressings soaked in solutions of carbolic acid.

Eclamptic or uræmic symptoms are among the other possibilities which may obtain during dropsy, and should be borne in mind. Uræmic attacks, with severe convulsions, should be treated, in strong patients, by venesection or by the energetic local abstraction of blood upon the forehead, whenever the prodromal symptoms (headache, vomiting, mild delirium, or slight coma) have been treated in vain, by means of baths with some rubefacient addition (for instance, caustic potash as recommended by Geissler), the promotion of diuresis, powerful drastics, cold affusion, cold to the head and præcordial region ; in mild uræmic conditions, with or without incipient convulsions, these latter means will generally suffice. Whenever the convulsions continue, in spite of free abstraction of blood, resort should be had to the inhalation of chloroform, the subcutaneous injection of morphine (especially in adults, and in any case in doses proportionate to the age), and to compression of the carotids.

SMALL-POX.

(VARIOLA, VARIOLOID AND OTHER MODIFICAITONS, BLATTERN, PETITE VEROLE, DIE POCKEN.)

CURSCHMANN.

Arch. Bd. 55 S. 545.—*Ponfik*, Ueber die anatom. Veränd. der innern Org. bei hämorrh. u. pust. Variol. Berl. kl. Wochenschr. 1872. Nr. 42.—*Zülzer*, Beitr. zur Pathol. u. Therap. der Variola. Berl. kl. Wochenschr. 1872. Nr. 51.—*Westphal*, Ueber Nervenaffect. nach Pocken. Ibid. Nr. 1.—Derselbe, Ueber eine Rückenmarkserkr. bei Paraplegie nach Pocken. Ibid. Nr. 47.—*Wendt*, Ueber das Verhalten des Gehörorg. u. Nasenrachenraums bei Variol. Arch. der Heilk. Bd. 13 S. 118 ff. u. 414 ff.—*Bierwirth*, Ueber Febr. varioloa. sine variolis, ibid. S. 226.—

E. Jenner, An Inquiry into the Causes and Effects of the Variolae Vaccinae, known by the name of the Cow-pox. London, 1798.—Idem, A Continuation of Facts and Observations of the Variolae Vaccinae. London, 1800.—*Sömmering* und *Lehr*, Prüfung der Schutzblattern durch Einimpfung mit d. Kinderblattern Frankf. a. M. 1801.—*Osiander*, Ausfürhl. Abhandl. über die Kuhpocken. Göttingen, 1801.—*Hessert* und *Pilger*, Arch. f. Kuh- oder Schutzpockenimpfung. Giessen, 1801.—A complete statement of the older literature is given by *Canstatt*, Med. Klin. See also the works of *Fuchs*, *Hebra* (Hautkrankh. Virch. Pathol.), *Wunderlich*, *Lebert*, *Barthez* et *Billiet*, *Trousseau*, *Rayer*, *Willan*, *Bateman*, etc.

HISTORY.

THE question as to the antiquity, spread, and origin of small-pox,[1] by far the most important of exanthematous fevers, has been for a long time the object of most zealous inquiry, without, however, eliciting an answer that can be designated as very definite.

It appears certain that small-pox is not indigenous in Europe. It reached us at rather a relatively late period from other lands, no one of which, however, can with certainty be alleged to have been its cradle.

The attempts which, with great pretensions to learning, have been made to show that Hippocrates, Celsus, and others were acquainted with this disease may be considered as unsuccessful, and even the assumption appears untenable that Europe harbored the evil guest at the time of Galen.

---

[1] The word "pock" has properly the meaning of "bag" or "sack," and therefore served originally to characterize the most prominent symptom of the disease, viz., the eruption. The word "variola" arose in a similar manner, being the diminutive of "varus," a pimple. It occurs in manuscripts of the ninth century, and originally included other skin affections accompanied by the formation of papules and pustules. Later the expression came to be applied to small-pox alone. The first physician who used it in this limited sense appears to have been *Constantinus Africanus*.

From remote antiquity, on the other hand, tolerably unambiguous accounts of small-pox have proceeded from China and Hindoostan; and indeed, according to Moore, its outbreak in these countries can be authenticated as far back as the year 1120.[1] Although, as Gregory, Friend, and Mead have shown, Moore's dates cannot be maintained with such precision, still, on the whole, we must share his opinion as to the great antiquity of the disease in those countries.

Many writers, Gregory for example, are inclined to regard as the first historically authenticated account of small-pox the celebrated description of Procopius (de bello Persico, lib. II. cap. 22) of a scourge which broke out at Pelusium about the year 544, and thence spread over Egypt, Syria, and the rest of Asia Minor. They base their opinion upon certain symptoms[2] mentioned by that author which are undoubtedly very well referable to variola. Others have considered the epidemic of Procopius as a mixed or transition form of the bubo-plague and small-pox; and believed themselves able to discover in its history grounds for this Darwinian representation. Both views are now entirely abandoned, and the epidemic is universally regarded as having been the true bubo-plague.

Not long after, however, in the year 581, we have an account, by Gregory of Tours, of an epidemic which, from the description of the chronicler, we must certainly recognize as variola. It raged in almost the whole of southern Europe, and was spoken of as "Lues cum vesicis, Pustula, Pustulæ, or Morb. dysentericus cum pustulis";[3] and Gregory distinctly separates it from the *Morb. inguinarius*, the real bubo-plague, which broke out at Narbonne in the year 582. It may appear strange that none of the physicians of that time even mention this no doubt long and destructive plague. But when it is considered how deeply the medicine of that period was sunk in superstition and mystery, and how entirely wanting was the desire for objective investigation, we shall wonder far less thereat, and shall prefer the simple record of the chronicler to any medical descriptions of that period.

---

[1] The statement is founded upon a work written in China at that time, "Treatise on Small-pox" (Teontahinfa), which contains a collection of the oldest medical knowledge of the Chinese concerning this disease. It is also reported from India that long before the birth of Christ a particular goddess had been worshipped as protectress against the disease, and also, that from a remote period her priests had practised the inoculation of small-pox for protection against the disease.

[2] Compare *Gregory*, l. c. page 34.

[3] Compare *Hecker*. Volkskrankheiten des Mittelalters.

To the Arabians, and among them to Rhazes, we are indebted for the first useful scientific accounts of variola.

Rhazes (900), who by the way ascribes to Galen a knowledge of small-pox, hands down to us, in addition, fragments of the pandects of Ahron (about the sixth century), in which this writer makes undoubted mention of the disease.

The descriptions of Rhazes are comparatively clear and perspicuous, and many of his therapeutic suggestions are highly judicious.[1] His theory of the disease, according to which small-pox is the expression of a ferment essential to all men, a boiling up of fluids impregnated with the small-pox virus from embryonic life, appears to us to-day paradoxical in its details.[2] The fermentation theory in general, however, has recurred from the Arabians, in the history of variola at all times, even the most recent (Liebig), merely modified in accordance with the medical views of the period.

Among Latin writers the already-mentioned Salernian, Constantinus Africanus, appears to have been the first to have given a scientific description of small-pox—in closest resemblance, it is true, to the descriptions of the Arabian physicians. Before Constantine little is known to us concerning the appearance of variola in Europe, except the aforementioned records of Gregory. That the disease occurred is beyond a doubt; but the limited means of communication at that time evidently prevented extensive spreading. At the time of the crusades we first see the scourge again assume terrible dimensions; just as epidemics in general cling to the heels of wars and great popular tumults. At the time of the crusades too, probably, small-pox houses were first generally erected in Europe.

---

[1] He ordered his patients, *e.g.*, to drink cold water, combated the abuse of purgatives, and, as a rule, treated light cases expectantly by suitable diet. He prescribed, too, steam-baths at the outset, and later, the inunction of oil and salt.

[2] The theory, moreover, is not peculiar to *Rhazes*, but is given, in its principles, by *Ahron* and *Mesue*. Even during the epidemic described by *Gregory of Tours* similar views prevailed among the people, whence the term "corales," then in use for the dreaded plague, which is said to be derived from the old German "koren," "kören" or "küren," *i.e.*, "to choose," "to separate," "to purify;" therefore indicating a sort of process of bodily purification from certain (corrupt) fluids. Bishop *Felix of Nantes*, as the writer relates, was a victim of this theory, inasmuch as, being attacked by the fever, he applied a cantharidal poultice to his thigh in order to hasten this excretion, and died of gangrene in consequence.

The disease effected an entrance into Germany towards the end of the fifteenth century;[1] while in England it had already been introduced in 1241–42. Sweden was visited by the scourge at a comparatively late date in the fifteenth century. Small-pox was conveyed from Europe to the American continent, where it does not appear to be indigenous, soon after the discovery of America. It first raged in Mexico, in the year 1527, and to a fearful extent, sacrificing its victims in millions; thence it gradually overran the whole of America. Later epidemics appear to have been repeatedly occasioned by the importation of negroes from Africa, the colored races in general being decidedly more liable to this disease than the white.

In the preceding remarks we have presented a few of the most reliable data in the defective history of small-pox, to the incompleteness of which numerous circumstances contribute. Not the least important of these is the fact that for a long time we did not know how to distinguish variola from other diseases. It was principally confounded with the plague, with other papulous and pustular cutaneous eruptions, and until a comparatively recent date it was invariably classed with measles.[2] Syphilis, also, to which the name "large-pox" had been assigned, was confounded with variola with special frequence; so that in England and France the latter was at an early date distinguished from the former as "small-pox" and "petite-vérole."

In Europe, as in other countries, small-pox long constituted one of the greatest scourges of mankind. Not a decade passed in which the disease did not decimate the inhabitants in one country or another, or over great tracts of country; so that it came to be more dreaded than the plague.[1] No matter how assiduously physicians labored to discover a method of treatment, none of them accomplished a result worthy of mention. The Arabian theories of the disease, requiring sweating and other remedies designed to "drive out the poison," were the order of the day till the first half of the seventeenth century. These notions, which still linger in the minds of the laity, however, were first overthrown by the talented Sydenham, who replaced them by the cooling, antiphlogistic method of treatment which we still consider rational in its fundamental principles. Yet, as may be imagined, even this treatment was but a feeble weapon against the terrible enemy. With the introduction of the inoculation of variola the great change first began in the history of small-pox. This was completed at a later date by Jenner's discovery of vaccination, and to-day the occurrence, intensity, and spread of the disease in different countries depend greatly upon the manner in which this eminently protective remedy is employed.

---

[1] Most probably imported from the Netherlands in 1493.

[2] *Sydenham* first definitely drew a sharp line between the two affections.

Starting from the knowledge that small-pox usually occurs but once in the same individual, the attempt had early been made, now and then, in Europe, to bring children intentionally into the vicinity of small-pox patients, in order that they might be protected for the remainder of their lives by means of a possibly milder form of the disease acquired in this manner. Actual inoculation of small-pox poison, however, was first practised in China and India, and even at a very remote period. It thence gradually extended through western Asia to Constantinople, where Lady Wortley Montagu learned the process, and promptly subjected her son to it (1717) with a satisfactory result. She carried the method to England, where she had her daughter

---

[1] In England, in the seventeenth and eighteenth centuries, seven to nine per cent. of all deaths were attributable to small-pox. In Berlin, from 1783 to 1797, one-twelfth of the total mortality, according to *Casper*, was due to the same. In the fourth number of the "Archivs der Aerzte und Seelsorger wider die Pockennoth," of the year 1798, *von Junker* computes the deaths occurring from small-pox in the year 1796 among the 7,000,000 inhabitants of Prussia at 26,646 (*Kussmaul's* letters). In the eighteenth century 80,000 died annually of small-pox in France. Of the resignation to the disease exhibited in the middle ages, a proverb of that time gives the best proof, "From small-pox and love but few remain free."

(1721) likewise inoculated; and she was soon enabled to bring the new process into widespread esteem, despite the violent opposition of physicians at the outset, and the silliest objections of the clergy. From England inoculation travelled rapidly to Germany, France, and other European countries; and the expectations to which it gave rise were actually realized in so far that the inoculated small-pox ran a decidedly milder course than the natural disease, and also afforded the same protection as the latter against new infection. The unfortunate feature of the method, and that which eventually condemned it, was, that individuals inoculated with small-pox were as likely to infect others as if they had contracted the disease naturally, so that provision was thus again made for the maintenance and spread of the variola contagion.

The introduction of vaccination put an end of course to inoculation; the merits of which it possessed in a high degree, without partaking of its disadvantages. A few words on the history of vaccination we reserve incidentally for a more thorough consideration of the subject.

ETIOLOGY.

Since the time of its first recognition variola has sooner or later found its way to nearly all the countries of the globe. Differing from most other epidemic diseases, small-pox exhibits a comparatively slight dependence upon conditions of soil or climate. It breaks out where predisposed individuals are exposed, under especially favorable circumstances, to the influence of its contagion. Certain geographical peculiarities of the disease have become greatly obscured since the general introduction of vaccination.

The disposition to small-pox is of very general prevalence. Individuals who can boast of an absolute immunity from the disease are quite exceptional.[1] In our day, when vaccination and revaccination afford such powerful protection against the disease, we have fortunately but limited means of observing the conditions of which the records of former centuries present to us such horrible pictures.

---

[1] *Morgagni*, *Boerhave*, and *Diemerbroek*, it is said, could claim this peculiarity, and the latter was evidently led from this circumstance to the opinion, which he upheld, that small-pox was contagious only in a very limited degree.

No period of life is wholly excluded from the susceptibility to variola. In this respect small-pox shows itself far more independent than the majority of acute infectious diseases, although a fully similar relation to all periods of life is in nowise to be asserted. In the earlier months of life the disposition appears to be less than after the first year. From that time until the age of forty years it is most strongly manifested, and tolerably equally throughout that period. Even from this age until sixty small-pox is frequently observed, and in the latest period of life its appearance is by no means a rarity.

The statement that children in the first months of life are less susceptible to the influence of small-pox poison than older children and adults, is found in the works of all the older authors. Even inoculation in these cases is said to be less effectual (*Moore*). Since vaccination has reduced the number of cases occurring in childhood to a minimum, we lack to-day the most important grounds for judging of this statement, viz., the opportunity for comparison.

Of the children who were born in the small-pox department, I did not see one that remained free from the disease, although a few, to be sure, exhibited but a strikingly slight development of the eruption. The following group of 632 cases will illustrate the susceptibility of the various ages (with the exception of children from 1 to 12 years), not one of whom, in Mayence, did I see taken sick after well-performed vaccination:

12 - 14	1	35 - 40	49
15 - 16	18	40 - 45	27
16 - 20	138	45 - 50	23
20 - 25	179	50 - 55	14
25 - 30	110	55 - 60	11
30 - 35	55	60 - 70	7

We see in this table the great participation even at a late period of life. The numbers between 50 and 60, and especially between 60 and 70, though small in themselves, express an enormous ratio when it is considered how relatively small a proportion of the population is composed of individuals of this age.

Even uterine life does not exclude the danger of small-pox infection, although the predisposition of the fœtus is far less than that of the child during the first few months after birth, and it may be included among rare occurrences for a child to come into the world already ill with small-pox, or with traces of having gone through the disease. Under what conditions an infection of the embryo takes place is wholly unknown. *À priori*, one would imagine that in consequence of the close relationship of its blood to that of the mother, the fœtus would pass through all the phases of the disease coincidently with her. Strange to say, however, this appears to have been observed but seldom. In the majority of reported cases the infection of the embryo appears rather to have taken place at a later period than that of the mother (pustular or desiccative stage), which would indicate infection by simple contact rather than by the blood. This is quite in accord with the assertion of older authors, that infection takes place most readily when the liquor amnii is present in least amount. Were the blood of the mother the principal infecting agent, the disease in the fœtus would be of decidedly more frequent, if not of constant, occurrence; for at no other time of life do such absolutely favorable conditions exist.

The following case will serve as an exemplification of the preceding argument:

M. G., a female servant, aged 22 years, in the fifth month of her first pregnancy, was sick in hospital with varioloid from November 28th to December 12th, 1870. The disease ran its course without complication. Fœtal movements during the disease were, just as usual, tolerably strong. On December 24th she was again admitted with acute gastric catarrh. On the 25th and the following days fœtal movements were at times very strong, and *on the 28th they suddenly ceased.* On the 31st, birth of a five to six months' child, evidently dead for several days (doubtless since the 28th), which presented *a well-formed small-pox eruption in the stage of suppuration*, covering the whole body; *least marked on the face*, and most abundant on back and buttocks. As far as this case goes, the period of infection of the fœtus does not coincide with the beginning, but with the latest period of the disease in the mother. The mother had fully recovered on the 10th of December, and could not have been the source of infection later. On the 28th, eighteen days afterwards, when the fœtal movements had finally ceased, the death of the fœtus doubtless occurred. If we accept for the fœtus the same duration of separate stages as is common to children under one year of age,[1] the appearances were such as to place the time of death in this case somewhere between the sixth and eighth day of the disease; and hence we must accept an incubatory period of at least ten to fourteen days, and the latest period of the disease of the mother as that of infection.

---

[1] It is scarcely possible to state exactly the duration and course of the separate stages in the fœtus. That the eruption is developed rather more rapidly than more slowly, and its stages completed, might be inferred from the circumstance that the fœtus is immersed, as it were, in a permanent warm bath, and in a condition very favorable to inflammation and suppuration.

Small-pox has been observed as early as the fourth month of embryonic life. I can vouch for its appearance at the fifth month of gestation from my own observation. Individual instances are cited in medical literature of perfectly healthy mothers (not attacked by small-pox during pregnancy) who have given birth to children affected with variola. It has usually been concluded that the embryo has been directly affected through exposure of the mother, who at the time was not personally susceptible to the contagion. The possibility of such an occurrence is not to be excluded. Some of these infrequent cases, however, might be explained upon the hypothesis that the mother suffered from "variola sine exanthemate," and thus infected the child. Such a connection would be the more readily overlooked, because the fœtus, as in the above case, does not suffer simultaneously with the mother, and the mild and rapidly progressing disease in the mother might be mistaken, or long forgotten before the birth of a child covered with the characteristic eruption. I am myself able to relate a case in point:

During a severe epidemic of small-pox, a midwife, aged 40, in the eighth month of pregnancy, fell sick with rigors, followed by violent fever, headache, pain in the back, etc.,—apparently the initial stage of small-pox. On the fourth day, however, she was free from fever, and, in spite of the most careful examination, exhibited no trace of the expected eruption. Ten days after the commencement of the disease, feeling at this time perfectly well, she gave birth to a child covered with a small-pox eruption, evidently just appearing, which developed still further, and in three days terminated in death during the stage of suppuration.

Sex, upon the whole, causes no difference in the susceptibility to the disease. Under similar conditions, men and women are liable to variola in the same degree. In women, of course, two physiological conditions, pregnancy and childbirth, occasion a certain predisposition to the disease, and also to its greater malignancy. This predisposition is counterbalanced in men, however, because their duties and employments far more frequently expose them to the danger of infection.

In regard to certain peculiarities of the several races of mankind, the dark-colored—and among these the negroes—appear to be decidedly more predisposed to, and on the average suffer more violently from, small-pox, than the white races; even under exactly the same conditions. Not only in their own country, but in foreign lands also, the blacks retain this peculiar sensitiveness to small-pox contagion.

The assertion is often read and heard that existing diseases lessen the disposition to small-pox; a proposition which is quite untenable in such generalization. The great majority of chronic diseases appear to exercise little or no influence upon the susceptibility to variola. We see patients, with heart, lung, and abdominal affections, attacked during an epidemic, as well as healthy persons, with only the fatal difference that they are more likely to fall victims to the scourge. In like manner lunatics, and patients with chronic cutaneous affections, are apparently no less seldom infected. Whether or not more exact statistical investigations would show any one affection, or group of affections, to be exceptional in this respect, we leave undecided. It would be an interesting problem at any rate to sift a good deal of material in this direction.

With acute affections, especially with the acute infectious diseases, it appears to be otherwise than with chronic diseases. As regards the acute exanthemata, especially scarlet fever and measles, their concurrence with small-pox was formerly much more frequently believed to exist than was really the case, inasmuch as the so-called prodromal eruptions, which will later be fully described, led not infrequently to an erroneous belief in such combinations. Indeed, even at a very recent date many authors[1] have not avoided this error. It would be going too far to consider all recorded cases of the simultaneous existence of small-pox and some other acute exanthem as such interchanges. At present we may consider as proven the possibility of the simultaneous affection of an individual with small-pox and with measles or scarlet-fever,[1] but we must regard the occurrence as rare, and in nowise consider the diagnosis easy. Especially should no one be misled (*e.g.*, by the lectures of Barthez and Rilliet) into the belief that the character of the eruption is in this regard the most reliable basis of judgment. On the other hand, far greater weight is to be placed upon other circumstances, especially the ætiology, the curves of temperature, etc.

---

[1] Compare, *e.g.*, the case of *Robinson* (Dub. Journ. of Med. Sci., May, 1872, p. 365, ff.), where hemorrhagic variola with a prodromal eruption simulating measles is described as a combination of measles with hemorrhagic small-pox.

A coincidence of variola and typhoid fever was not established beyond a doubt until a most recent date. But the possibility of this occurrence, especially since the publication of the decisive case of Th. Simon,[2] is not to be denied.

Apart from these rare occurrences it can be asserted with certainty, *that for an individual suffering from scarlet fever, measles, or typhoid fever, there is, during the entire duration of the affection, a very slight liability to an attack of variola only.* There is a certain exceptional proportion in this relation.

In the hospital at Mayence, where the small-pox building stands very near the other wards, I had the opportunity of observing with unfortunate frequency the relation of patients otherwise affected, particularly those attacked by typhoid fever, to the small-pox contagion. Typhoid-fever cases are hardly ever wanting in the hospital at any period of the year; yet during the entire duration of the small-pox epidemic, almost two years, I saw no case in which infection had taken place during the course of the typhoid processes. I saw, however, unfortunately, a considerable number of typhoid convalescents attacked. Reckoning back in these cases from the beginning of the first stage of variola, the longest possible accepted period of incubation, the date of infection always occurred at the time of complete convalescence, *i.e.*, at a period when the body temperatures had permanently returned to the normal degree. Among six infected typhoid convalescents, of whose cases I have special notes, the primary stage began in two cases on the nineteenth, in two on the seventeenth, and in two others respectively on the sixteenth and fourteenth day after the last febrile temperature (referable to the typhoid fever). Hence, even in the last case, supposing a period of incubation of at least fourteen days, the date of infection would fall upon the first day free from fever.

---

[1] Compare *Körber*, Petersb. Zeitschr. XII., *Steiner*, Jahrb. für Kinderheilk. Bd. I. 4 (Morbill.-Variola), *Fleischmann*, Zeitschr. für Dermatol. u. Syph. IV. Jahrg. ditto Jahrb. für Kinderheilk. Bd. IV. 2 (Scarlat.-Variola).

[2] Berl. klin. Wochenschr., No. 11, 1872. The case was observed during the simultaneous existence of an epidemic of typhoid fever and small-pox. S. appends a note of remarkable value, considering the great reliability of this author, that in many cases of typhoid fever a more marked development of the roseola became noticeable at that time (under the influence of small-pox).

The cases appear to me to be especially instructive, in so far that—as could not be otherwise in an overcrowded hospital during a violent epidemic—external opportunity for infection being present as much during the typhoid fever as after it, the opportunity was afforded of seeing the same individuals exposed to tolerably similar contagious influences in the typhoid and in the relatively normal condition.

The assertion of many authors, that influenza (Rosenstein), malaria, and whooping-cough lessen the possibility of infection with small-pox, I will not pass over in silence, although recent literary data or personal observations in the matter are not at my command.

As we have just seen, in regard to certain diseases, so also observations relative to the infection of healthy persons may be cited, which can scarcely be otherwise explained than upon the assumption of *a temporary failure, or at least an occasional diminution of the susceptibility to small-pox contagion.* It is not infrequently noticed that in healthy persons, exposed in tolerably equal degree during long continuance of the prevalence of the contagion, when finally attacked, the period during which infection could have taken place is longer than the longest possible normal duration of the stage of incubation. Such observations are made with more precision in unvaccinated individuals, since in the case of those attacked after vaccination or revaccination the objection (though perhaps rather far-fetched) may be raised that we do not know but that the person was still under the protecting influence of the vaccine up to the very day of infection.

The history of small-pox inoculation presents us with data concerning the temporary insusceptibility to contagion, which have almost the convincing power of experiment. We learn that this operation (performed with the same lymph and under similar circumstances) by no means produced the desired result in all cases. Gregory (l. c., p. 19) states that Woodville reckoned the proportion of temporarily non-susceptible children to be 1 : 60, and of adults 1 : 20.

In the case of most persons the disposition to small-pox, after one attack, disappears for the remainder of life, or at least for a long time. We have as yet scarcely an idea of the relations of this strange manifestation, since we still lack, as we shall see, an accurate knowledge of the nature of the small-pox contagion, which is the *conditio sine quâ non* of its explanation. When, twenty-five years ago, the resplendent light of the newly arisen organic chemistry threatened to obscure all other scientific methods of research, it was hoped that by its aid the problem would be solved; but we will not reproduce here those long-abandoned misconceptions. Exceptional cases have occurred where individuals have been attacked with small-pox for the second time, and even since the introduction of vaccination. Indeed, persons have been reported so unfortunate as to contract the disease more than twice, even five and six times.

The lapse of time between such recurrences is reported as differing widely; ofttimes as extremely short. An intermission of a few months constitutes the extreme of brevity, while more frequently there is an interval of from two to three years.

Extreme caution is requisite in accepting such "secondary or recurring" small-pox, and all acounts of this kind are not to be taken for granted. It is seldom that the same physician has himself observed bôth or all attacks, which would be the surest protection against error. To rely upon the statement of patients who may characterize all imaginable skin eruptions as small-pox, is entirely out of the question. Had not the individual credulity of observers come into play there could not exist, even taking into account the intensity and character of the epidemics, such entirely irreconcilable numerical statements concerning the relative occurrence of secondary small-pox. These statements vary between 1 : 250 (Eichhorn) and 1 : 10,000 (Condamine).

It is said that in general a violent attack of small-pox is a better protection against future attacks than a mild one; and for extreme cases this statement is perhaps true. But that even the slightest infection in this respect works favorably, was proven by small-pox inoculation, the mother progenitor of the beneficent vaccination. It has been repeatedly asserted that when small-pox attacks a person for the second time, it generally runs a milder course.[1] Yet exceptions to this rule occur, as is illustrated by the well-known case of Louis XV. of France, who, after having variola in his fourteenth year, died of the same disease in his sixty-fourth.

---

[1] *Hebra*, on the contrary, holds that the prognosis in the case of those attacked the second time is especially unfavorable.

It is asserted on the part of many (Gregory) that certain conditions—puberty, inflammatory diseases, changes of climate, etc. —favor the reawakening of the predisposition.

Of the earliest origin and mode of extension of small-pox the most varying opinions have prevailed since the disease first became known, in accordance with the varying theories as to its nature. The most manifold general causes—certain conditions of the earth or of the air, changes of temperature, moisture, etc., were formerly held responsible; and the possibility of a miasmatic origin of the disease was never for a moment doubted. Boerhave first overthrew these ideas, and established the theory of the exclusive spread of variola by contagion, which to-day we regard as firmly grounded and may formulate as follows:

Small-pox spreads at the present time exclusively by means of a specific virus, which is begotten in the body of a small-pox patient, and conveyed directly or indirectly to a predisposed individual, thus causing the outbreak of variola again in the latter. Though here and there one still reads or hears that at least mild "incomplete" forms may arise without contagion, no reliable proofs therefor can be adduced. These statements are based upon the very frequent failure to trace directly the source of contagion; a difficulty resulting from certain peculiarities to which reference will subsequently be made.

Of *the nature of the small-pox contagion* there is, so far, nothing definite known. On the other hand, tolerably accurate knowledge is possessed of some of its peculiarities. These we will pass in review, and ascertain to what extent, from its effects, conclusions may be drawn as to the agent.

The contagion reproduced by an affected organism *takes effect first of all upon the patient himself.* It is principally the small-pox pustules which contain this, as is clearly proven by small-pox inoculation. This teaches us further that the virus is most active at the time when the previously clear serum of the pustules begins to become turbid. That inoculations succeed in the later stages also, is sufficiently well known. Even the small-pox crusts possess the power of infection in so high degree that they were even used for inoculation.[1]

Whether the secretions of patients (when small-pox pus or crusts are not accidentally mixed with them) are infectious when inoculated upon other individuals is doubtful; according to the direct experiments of the older authors perhaps the question is even to be answered in the negative. Such experiments have been made with urine, saliva, expectoration, and even with fæces.

In like manner blood inoculated from man to man is said to be altogether ineffective. I consider this as at least doubtful. Direct inoculatory experiments upon men, upon which earlier observers relied, are regarded as absolutely criminal in the present day, since inoculation has been supplanted by vaccination; and therefore, unless proved by accident, we cannot expect any solution of this problem. Certain experiments upon animals, therefore, appear the more significant in reference to this question. Osiander was able to inoculate healthy sheep successfully with the blood of other sheep affected with small-pox. Still more important is the experiment of Zülzer, who inoculated a monkey with small-pox with the blood of a variolous patient.

The contagion not only adheres to the patient himself, but *is also present in his immediate vicinity, in the "perspiration" impregnated with transportable gaseous particles.* It appears to be "secreted" by all emunctory surfaces of the body, among which the skin naturally plays a prominent part. Whether the frequently noticeable peculiar (specific?) odor of the patient is closely related to the contagion, as was formerly believed, is quite undecided. In general it can be said that the contagion loses efficacy with increasing distance from the patient. Predisposed persons are the more readily infected the longer they remain in the vicinity of patients, the smaller the apartment, and the greater the number (and severity) of the cases occupying it. In large spacious apartments, with few patients or but one, the danger is slighter; and it is still more decreased in the open air.

---

[1] The crusts were formerly used in China in the most singular manner, inoculation being practised by placing them in the nose. Even in more recent times, powdered small-pox crusts were recommended, *e.g.* by *Gatti*, as convenient inoculatory material.

It is difficult to determine with accuracy the *stage in which patients are most likely to spread infection* (by diffusing the poison through the neighboring atmosphere); the earliest period of suppuration is usually so considered. Many ascribe an especial danger to the stage of desiccation; but on insufficient grounds, however. When there has been frequent communication with patients, the time of infection cannot be accurately determined, principally on account of the variable duration of the incubatory stage.

In the initial stage, moreover, at a time when as yet no trace of small-pox eruption exists, *infection may take place;* and infection is also possible during the period of incubation, which is generally free from every symptom of the disease.

The frequent cases in which patients are unable to indicate the source of infection, not having come in contact with any small-pox patient, are in part to be referred to infection during the initial stage.

The possibility of transferring the disease from an individual during the period of incubation is exemplified in an interesting case reported by Schaper (Deutsche Militairärztl. Zeitschr., 1872, p. 53): In the Charité Hospital of Berlin, small pieces of skin were taken for transplantation upon other individuals from the amputated arm of a person who, before and at the time of the amputation, did not manifest the slightest symptom of general disease. Several hours after the amputation the patient was attacked with violent fever, followed two days later by an eruption of small-pox. One of the individuals upon whom the transplanted skin had been placed was attacked by variola on the sixth day after the operation; the three others remained exempt.

Besides the atmosphere about the patient, *the contagion is imparted to certain objects which have been used by him or been in his neighborhood; and it clings to them also for a variable length of time after they have been removed from his personal influence.* Foremost in this respect may be mentioned clothing and bedding; and especially all things which have a rough, woolly surface. According to general belief, the poison adheres much less, or not at all, to smooth objects, especially glass, metal, etc. (dried small-pox secretion, of course, being excepted).

The personal effects of a patient are not alone able to convey infection, but *healthy persons also, under some circumstances, carry the poison in their clothes ;* so that caution in this respect is especially necessary on the part of physicians, nurses, etc.

Small-pox virus impregnating gaseous media, and excluded from the air, is possessed of very great permanence. Where this exclusion has been as complete as possible, the duration of contagiousness of the virus has been estimated at many months, and even years, without, however, adducing exact evidence for the extremest statements.

Exposed to the atmospheric air the contagion is readily destroyed ; most probably by being more and more diluted in the air until it becomes inefficacious and finally disappears.

High temperatures, vapors of chlorine, iodine, bromine, and sulphur, and alcohol, act deleteriously upon the contagion, though only after very intense and long-continued influence.

Infection may undoubtedly take place through the medium of the bodies of patients dead of small-pox. Whether a reproduction of the contagion may follow in them, is hardly to be determined in our ignorance of its nature. It is more probable that it is the same thing with cadavers as with other life less objects, the virus generated during life simply adhering to them.

Concerning the mode in which the contagion adherent to transportable gaseous particles enters the body in case of infection, we still lack accurate knowledge. According to a general and quite probable view, the poison is principally absorbed, in breathing, by the mucous membrane of the respiratory tract. That this may happen in the upper portion of this tract—the nasal mucous membrane—is evident from the ancient method of inoculation by the introduction of the crust into the nose. We may look upon the exceedingly fine particles detached from the skin of the patient, and from the pustules and crusts, and which are suspended in great numbers in the air surrounding small-pox patients, as the carriers of the "inspired" contagion.

It does not follow, however, that this particular mode is the only one in which the respiratory mucous membrane absorbs the contagion. It may not even be the most important.

The possibility of the absorption of the virus by the mucous membrane of the alimentary canal, especially by the stomach, is positively asserted by the older writers. Indeed, it was regarded as quite an ordinary means of infection. Camper assures us that he observed the outbreak of severe variola in a patient who had swallowed some small-pox pus; and it is said that it was formerly the general custom in Bengal to inoculate variola by the deglutition of small-pox crusts (Eimer). Very recently, Zülzer met with a negative result in the case of a monkey who was made to swallow the crusts. The question is not yet to be considered as definitively settled.

It is highly probable that the contagion never enters the system through the external unbroken skin. Both at an early date, and more recently, experiments have been made to determine this point, and wherever a success has been noted, it has almost always appeared that the virus had been rubbed into the skin with force, and for a length of time. It is probable, therefore, that the skin became superficially wounded in the operation.[1]

We have thus far seen, that, concerning the majority of questions relating to the origin and spread of small-pox, only so much as is general is known with certainty. Mystery and doubt prevail wherever we investigate more closely. The principal reason of this lies in our ignorance of the primal causes of the entire process, and especially of the nature of the contagion. Positive views and ideas concerning these questions are to be found indeed from the earliest accounts of variola; but these have undergone constant change, chiefly in accordance with the changes of pathological ideas.

The Arabians believed, and Rhazes expressed the decided opinion, that the small pox process was derived from the fermentation and effervescence of a certain material present in the blood of every person from the embryonic period. Sydenham and von Diemerbrock likewise held the opinion, that in the period of incubation, a kind of fermentation took place in the blood, the product of which was manifested in the outbreak of small-pox. This theory of fermentation held its ground from that time until the first half of the present century. It gained fresh support again with the development of organic chemistry under Liebig, who merely specialized the old views and revived them in accordance with the ideas of modern chemistry.

---

[1] In India, where small-pox inoculation has been practised by the Brahmins from a very remote period, this result was accomplished, as we are told, in the very manner indicated,—by the application of cotton saturated in small-pox pus upon a portion of the forearm previously made sore by rubbing.

Within the last decade these theories have gradually been losing ground. Following in the progress of the times, views as to the parasitic nature of the contagion have taken their place;[1] and these are now advocated with the same confidence with which the supporters of the chemical theory formerly stood up for their opinions. An opportunity will be offered in the anatomical part of this article for a full consideration of these questions. We will simply remark here, that we are still very far removed from their solution, and prefer to advise an especial skepticism in this matter, without maintaining an attitude of positive opposition. Even the most practical observers are in danger of being borne along the rapidly swelling current of modern parasitic research, which, without doubt, conceals some golden grain.

In concluding our ætiological observations, let us say a few words concerning the general *conditions favorable to the spread of the disease.*

Small-pox is seen in all possible grades of intensity and extent, from sporadic, domestic endemics, or small epidemics, to the fatal ravaging scourge which overruns entire countries, and is not checked until it has attacked nearly all who are predisposed. In the larger cities of Europe small-pox is seldom at any time entirely extinguished. Isolated cases are almost constantly observed there, like a lingering, slumbering fire that bursts forth in mighty flames from time to time, under a combination of favorable circumstances. As in frequency, so also in intensity, small-pox exhibits the greatest variation in individual endemics and epidemics. That its danger has diminished a great deal, on the whole, since the introduction of vaccination, is beyond the slightest doubt. Nevertheless we pass through epidemics in Europe (as for instance that of 1870–71) which carry off from eighteen to twenty-five per cent., and even more, of all attacked.

---

[1] *Nil novi sub sole* applies also to this theory, although apparently so modern. As early as the seventeenth century some (*Chr. Lange, Borelli*) believed that they had seen worms in the small-pox pustules. In the present century, *Henle* in particular has advocated with great energy the parasitic nature of certain diseases, including small-pox, in opposition to the chemists. *Gluge*, also, as early as 1838, described small shining bodies, often united in small chains, as integral elements of small-pox lymph.

The attempt was formerly made to prove that small-pox epidemics return with mathematical accuracy at certain intervals.[1] The truth at the base of this exaggeration is the simple observation that, from an early date up to the present time, fluctuations have occurred in the recurrence of the disease, so that periods of rest have alternated, with some uniformity, with years of intense prevalence. Although we are not yet perfectly clear in our ideas respecting this sort of periodicity, still certain facts may be advanced which influence it at the present time. We know that one attack of small-pox protects a person for life, or at least for a long time ; and that the inoculation of cow-pox exerts the same effect also, although it is of shorter duration. In a population, then, where as many individuals as possible are subjected to these favorable influences—and in vaccination we have a sure means for its agreeable accomplishment—the chances of the disease taking a firm hold and spreading become extremely slight. But when this immunity has lasted a certain time, and the inoculation of cow-pox has therefore been insufficiently carried on, then, of course, the susceptibility of those previously protected becomes gradually re-established. To this number still others are gradually added, who have neither passed through the disease nor been vaccinated. In this manner the fruitful soil is again provided upon which the contagion only needs to work to give speedy rise to a scourge of mighty dimensions. If such an epidemic is finally extinguished, all the predisposed among the community have usually been attacked, and thereby become exempted again for a long time. Moreover, the second important factor, vaccination, is more carefully performed under the fresh influence of fright which attends the return of small-pox ; and this likewise protects for a long time afterwards. Hence a long time must elapse before available material has again been collected for the disease.

---

[1] A recurrence of the disease every twelve or fifteen years has been stated.

PATHOLOGY.

A few preliminary words only are necessary concerning the limitation and division of the subject. Complete accord prevails, at the present time, in reference to the relation of *variola vera* to varioloid. No one any longer considers varioloid as an independent disease, as was formerly zealously maintained. Every one knows that we see nothing further in this affection than one of the many forms in which small-pox is manifested according to circumstances. All the remaining "forms" of variola, which we shall study more closely as we proceed, are to be regarded in the same light.

Concerning the relation of varicella to variola, no perfect unity of opinion has yet been reached. While Hebra's view of the close connection of both processes was universally respected until a short time since, and has its supporters even at the present day, authoritative voices are again raised in favor of their separation. In this manual, firm ground is taken for the view that varicella is not a simple modification of variola, but a disease *sui generis*. The section relating to the disease in question is written by the most prominent champion of this view ; and we will content ourselves here with simply referring to his arguments.

Symptoms and Course of the Disease.

Stage of Incubation.—From the moment of reception of the variola contagion until the consequent outbreak of the disease, a certain time always elapses during which the individuals, as a rule, present no abnormal symptoms, subjective or objective ; or merely make some vague, insignificant complaint. This period we term the stage of incubation. Concerning its duration in small-pox we are better informed than in other acute exanthemata, for the reason, perhaps, that it exhibits greater regularity in this respect.

In the cases where I have been able to fix the duration exactly, I found it, in accordance with the majority of authors, to be most frequently from ten to thirteen days, less frequently fourteen days or eight to ten days; and in one case only five days.

After inoculation of variola, forty-eight hours elapse before the commencement of the resulting phenomena.

A frequent opportunity is by no means presented to determine accurately the period of incubation. (I was able to do so but ten times in 1,034 cases.) Those cases only are available in this connection in which the individual *is known* to have been exposed to the contagious influence *but once, and for a short time.* Where exposure has taken place repeatedly, or for a long time, correct data for the computation are evidently lacking. I would not care to rely, without further examination, even upon observations which satisfy this requirement, provided they were made during a great epidemic; since, in addition to the single authenticated exposure, the individuals may, unknowingly, have been repeatedly exposed to contagious influences (through healthy persons, articles belonging to patients, patients in the primary stage, etc.). Perhaps many of the cases of unusual length or brevity of this stage, sometimes occupying several weeks, or, just the reverse, a few hours only, may be explained by such oversight.

We do not know whether the manner of infection or certain peculiarities of predisposition play a part in the duration of incubation. We must not omit to mention that Zülzer found the stage of incubation in hemorrhagic variola to be considerably shorter (six to eight days in nine cases) than in pustular variola.

Whatever may take place in the body during this period, when the infected person ordinarily presents nothing abnormal, is yet unknown to us. The contagion, of course, does not slumber; certain processes go on, rather, in the meanwhile, which do not yet give rise to distinct symptoms. In this very connection, however, it is interesting to note that in rare cases certain vague symptoms of disease, such as languor, pain in the head and back, or slight gastric disturbances are observed, even during the period of incubation.[1] Sometimes, indeed, a pharyngeal catarrh, with redness and swelling of the uvula and tonsils, has been noticed in the last days of this stage (Obermeier).

---

[1] With almost all of my own patients, from whom a regular history could be obtained, I have sought on theoretical grounds for some symptoms during the stage of incubation; but out of 1,000 cases, in eleven only (about one per cent.) could I authenticate such symptoms with certainty. *Scheby-Buch* saw the stage of incubation accompanied by symptoms in about four per cent. of all his cases. I am inclined to regard this large proportion as a peculiarity of the Hamburg epidemic, and believe that my own figures come nearer the rule. *Buch* was unable to discover any prognostic indication in the appearance of such symptoms, an observation which I can substantiate as far as my own few cases go.

Initial Stage.—Only in the most infrequent, and then mostly in very mild cases, does the characteristic exanthem make its appearance immediately upon the termination of the period of incubation. As a rule, the outbreak of the eruption is preceded by a more or less considerable space of time, usually associated with fever and disturbance of the general system, which we will designate as the *initial stage.*[1] The transition of the stage of incubation into this new phase of the disease is a gradual one only in the minority of cases; and then most frequently in those in which the stage of incubation was not free from symptoms of disease. Almost constantly the beginning of the initial stage can be fixed with a certain accuracy. Its duration, the termination of which is well marked in the outbreak of the eruption, is tolerably constant. As a rule, it lasts three days; although from two to four days are likewise not unusual. A still shorter or longer duration is far more rare, and probably indicative of varioloid; which, in respect to the extent and nature of its initial stage, exhibits far greater variations than true variola.

In forty-two per cent. of all cases in which I could limit the initial stage accurately, it amounted to three days; this being, in variola vera, almost a rule. A shorter duration of this stage was decidedly more frequent than a longer one; a duration of from one to two and a half days being found in thirty-eight per cent., and one of four days or more in only ten per cent. of the cases examined in reference to this inquiry. Sydenham at times saw the initial stage lengthened to six or seven days; and de Haën indeed speaks of fourteen days. That the initial stage is usually lengthened in *Febr. variol. sine exanth.*, and in *purpura variolosa*, we shall have occasion to mention hereafter.

As for the influence of age upon the duration of the initial stage, it appears to be somewhat shorter in children than in adults. Barthez and Rilliet found two days to be the most frequent duration. In old age, again, the course of this stage is retarded, just as in certain chronic diseases.

---

[1] Although no admirer of new nomenclatures, I have, nevertheless, in accord with *Canstatt* and *Obermeier*, thought it best in this article to drop the expression "Prodromal stage," heretofore in use, which involves false ideas, and to take the liberty to replace it by the simple and less pretentious term of "Initial stage." The old, incorrect designation took its rise, as is well known, from the fact that the imposing eruption was instinctively conceived to be the essence of the disease, and not simply one of its most important localizations. As is generally known, it may be entirely wanting (*Febr. variol. sin. exanth.*), and it is indeed absent in purpura variolosa, the most dangerous form of the disease.

To draw from the duration of the initial stage precise conclusions respecting the intensity of the subsequent stage, and of the course of the disease in general, I hold to be unjustifiable in spite of contrary assertions made here and there, chiefly upon the authority of Trousseau. The passage in question in Trousseau reads:[1] "The longer the manifestation of variola upon the skin is delayed, the more harmless will be the affection; but inversely, likewise, the more rapidly the eruption is developed, the more dangerous will be the disease." In what manner we are to explain the origin of this view, whether, as would seem most likely, through the changeable peculiarities in the character of the several epidemics, we leave undecided.

We notice a far greater variation in regard to the *intensity* of the initial stage than in its duration. We find this stage manifested in all possible grades and under the most varying forms, from slight and often wholly overlooked disturbances up to the severest picture of the disease, attended with extremely violent fever. Isolated cases of death have been recorded in almost all extensive epidemics, fatal before a trace of eruption had appeared.

How these differences are occasioned is not yet accurately known; but individual peculiarities appear to play no inconsiderable rôle. It can in nowise be maintained as a general rule that the intensity of the initial stage bears a ratio proportional to the severity of the entire morbid process. The most violent symptoms, especially in sensitive individuals—women and children—are not infrequently seen to eventuate in a quite harmless varioloid. With a mild onset of the initial stage it may be more justly predicted that severe variola will not follow, or only as a rare exception.

The usual symptom which distinctly marks the beginning of the initial stage is a *violent rigor*, or, as I have observed more frequently, several chills, usually of slight intensity, repeated at fixed intervals. Fever is thus ushered in, usually lasting until the eruption appears, and in isolated cases of varying intensity; but sometimes so severe that in conjunction with the other symptoms a picture of disease is presented severer and more serious than almost any other to be witnessed in acute affections.

---

[1] Med. Klin. II. Aufl. German by *Culmann*, Vol. I., p. 5 and 6.

The temperature of the body often rises on the first day to 103° or 104° Fahr., and subsequently with comparatively slight morning remissions, and much more rarely discontinuous, rises considerable higher, so that on the evening of the second or third day it frequently rises to 105° or 105½° Fahr., and in some cases, not the most infrequent, even above 107° Fahr.[1]

The *pulse*, usually full and tense in the previously healthy individual, generally corresponds in frequency with the temperature of the body, so that the curves of both run approximatively parallel. Even in full-grown, powerful men, it is seldom under 100; usually it is from 108 to 120; in women frequently 130 to 140; and in children as rapid as 160. In very rare, and principally severe cases, I found the pulse soft and sensibly dicrotic, resembling the condition so frequent in typhoid fever.

The frequency of respiration is almost always considerably increased. The respirations are short and labored; and many patients actually complain of dyspnœa; although the most careful examination reveals no essential change in the respiratory or circulatory apparatus. I must say, that to my mind the repression of the respiration stamps the picture of a severe initial stage as somewhat characteristic, and it gives me the impression that its frequency often reaches a somewhat higher degree than in other febrile systemic affections with a similar temperature of the blood. Perhaps this is explicable upon the ground that in few febrile conditions does the temperature of the blood rise so quickly from a normal to the highest degree, and that just in this rapid manner of ascent there is an additional essential element, namely, the irritation of the febrile heat upon the respiratory centres.

---

[1] These figures, like nearly all the observations of temperature in this treatise, are those obtained in the axilla. Sphygmographic observation, which I frequently made, furnishes, as might be expected, nothing characteristic, but rather the same curves as are produced in analogous acute general febrile disease.

The patients are languid and weak in proportion to the grade of fever, and even the most powerful individuals are hardly able to stand out of bed. While, for example, typhoid-fever patients among the lower classes, with temperatures already quite marked, are able not infrequently to make their way to hospital on foot, patients in a severe initial stage of small-pox stagger as if drunk, and are scarcely able to take a few steps without support. When observed out of bed the face often appears sunken and pale, the features expressionless, the extremities cold, the arterial vessels contracted, and the pulse small and extremely frequent; so that many patients, when seen soon after in bed with a red turgescent countenance, are hardly to be recognized.

In the majority of such cases the skin feels hot and dry, or is covered with a moderate perspiration. Profuse perspiration continuing through the following stage, as generally witnessed by Trousseau in the epidemics which he observed, and which he was inclined to regard as a favorable critical event, occur much more rarely.[1] According to many authors, and particularly the older ones, it is asserted that this perspiration already possesses a specific odor; a fact, if so, of which I have not as yet been able to convince myself, in spite of tolerably delicate olfactories.

Most patients complain of severe thirst. The lips and tongue appear much parched. The tongue is usually covered with a thick, whitish-yellow coat, and an insufferable *fœtor oris* is sometimes already noticeable.

The *appetite* is almost always entirely in abeyance. At the same time nausea, gagging, *and actual vomiting* are frequent as distressing symptoms. While in most cases vomiting is present only at the beginning of the initial stage, it continues, in others, with great obstinacy through its entire duration, and is often bilious. In cases where hemorrhagic forms are subsequently developed, vomiting and gagging are frequently as obstinate as in toxic gastritis, or even in obstruction of the bowels. I recollect one case of rapidly fatal hemorrhagic variola, which was sent to the hospital in the initial stage with the apparent diagnosis of intussusception. In addition to the vomiting, complaint is often made of violent pain and dull pressure in the epigastrium, and these symptoms appear to be especially constant in hemorrhagic variola.

---

[1] In the Hamburg epidemic of 1871, also, this profuse sweating was not so frequently observed. Compare *Knecht*, loc. cit., p. 167.

Most patients suffer from *constipation*, which often continues during the entire course of the disease, and requires therapeutic interference. Diarrhœa without other apparent causes is rare in adults, though somewhat more frequent in children.

Headache is among the most constant symptoms, and scarcely ever entirely absent. It appears, as a rule, shortly after the chill, or simultaneously with it; or it may precede this a few hours, associated with pain in the back, languor, etc. It either continues unchanged during the whole initial stage, or, as is more common, gradually subsides as the eruption approaches. Meanwhile the headache is unusually severe, often to such a degree that even powerful men, usually of great endurance, are forced to make loud outcries. Its usual location is the entire head, and when a particular limited spot is designated, it is usually referred to the forehead. Patients describe this pain in the most varying manner; as if a rope tightly encircled the head, or as severe lancinating, throbbing pain, increasing with every pulsation. The face is usually red and bloated, the forehead hot, and the carotids in violent pulsation.

Towards evening, especially on the second or third day, it is not rare to hear patients *talk incoherently* now and then; and many even fall into violent *delirium*, which cannot always be considered as alcoholic, although that form may certainly be included among the most frequent occurrences. Nearly all patients suffer from sleeplessness and great disquiet.

Coma I have seen but once in the adult (in a case terminating fatally in the initial stage), but often in children. *Convulsive symptoms* are sometimes associated with it, sudden starting-up, grinding of the teeth, and even complete "epileptiform" paroxysms (Sydenham, Peter Frank, Trousseau, *et al.*). Sydenham found this to occur more frequently than before the outbreak of other acute exanthemata; and the observation appeared to him of diagnostic value.

Many patients complain of *vertigo;* and in sensitive individuals *syncope* often occurs upon attempts at rising. Even collapse is now and then observed, and is generally of evil augury. ***Pain in the back*** is a no less striking symptom than headache, and generally seems to be especially characteristic of the primary stage of small-pox. It is by no means as constant as the gastric symptoms and the headache. I have observed it in rather more than half my cases, and in one-third it was so severe that the patients voluntarily complained of it. It appears as though the pain in the back were found more frequently in variola vera than where varioloid is subsequently developed. It is most constant and violent in those cases which become hemorrhagic.

Like the headache, this symptom usually appears just at the commencement of the initial stage, sometimes a few hours before the chill, and continues until the outbreak of the eruption. The seat of pain is usually in the lumbar region, extending down to the sacrum, and at times confined to this region, while it is less frequently complained of higher up towards the dorsal vertebræ. Stiffness and contraction of the muscles of the neck, on the contrary, occurs somewhat more frequently. When pain in the back is present, it constitutes one of the principal troubles of the patient. When accompanied, moreover, with *drawing, tearing pains in the extremities*, as is very often the case, it may be erroneously attributed to acute articular rheumatism, especially in sporadic cases, or the first cases of an epidemic. In pregnant women the pain in the back may be mistaken for labor-pains, and in those not pregnant it is often simply regarded as the precursor of the speedy appearance of the menses.

Bronchitis occurs among the *less constant symptoms*, and is far more frequent in the stage of efflorescence. In patients previously suffering from pectoral affections there is usually an exacerbation of their symptoms, cough and dyspnœa ordinarily undergoing increase, and pleuritic pains being increased or appearing anew.

No important anomalies affect *the heart*, if this organ has been previously unaffected. In the case of sensitive females and topers palpitation may occur now and then, although without perceptible material occasion for it.

Not infrequently *anginose symptoms* occur towards the end of the initial stage. Swelling and diffuse redness of the tonsils and soft palate are usually apparent at that time, and, much less frequently, discrete red spots are already apparent upon these parts. Most frequently the throat affections occur in cases in which definitely grouped pustular eruptions are established, later, upon the mucous membrane of the mouth and pharynx. Coryza sometimes appears in connection with the anginose symptoms, and with this epistaxis, photophobia, and lachrymation may also be associated. In other infrequent cases the affection of the mucous membrane extends down into the larynx, so that the various degrees of hoarseness are produced.

I have directed my attention to *the condition of the spleen* in a large number of patients in the initial stage (and also during the subsequent course of the disease) without, however, having arrived at any definite results. So far, I only feel safe in saying that I have never been able to detect an enlargement of the organ in the initial stage of varioloid; while this has frequently occurred to me, and often in a marked degree, in true variola. In other cases of variola vera, however, the splenic tumor in the initial stage has been wanting. I have not succeeded in establishing any law in reference to its presence or absence. The condition of the organ in severe grades of the disease, however, appears to me to be well deserving of study; and more extended observations on the occurrence, beginning, and duration of the splenic enlargement during future epidemics would prove, perhaps, a task well worth undertaking.

When certain authors speak of an *enlargement of the liver* as belonging to the initial stage, it is, perhaps, theoretically deduced from certain post-mortem appearances, to be subsequently, described. Here and there, indeed, I have found the hepatic region somewhat more abnormally sensitive on pressure, but never distinctly enlarged as a result of the acute process.

In the initial stage the examination of the *urine* does not yield anything characteristic. The urine is usually tolerably concentrated, according to the degree of fever; and the quantity passed in the twenty-four hours is diminished. The chlorides are usually considerably diminished, sometimes to a minimum; and the other solid constituents are quantitatively proportional to each other, and to the degree of concentration. In severe cases more or less albumen is frequently to be detected in the urine, sometimes more than is usually found in similar grades of fever from other causes. Grounds explanatory of the last symptoms are to be found in the anatomical portion of this article. Abundance of albumen is an unfavorable prognostic symptom. In cases which terminated in hemorrhagic small-pox I found albumen tolerably constantly and early. Women not infrequently complain of strangury, but I do not recollect this to ave been the case with men.

Special attention has recently been very properly directed to he *condition of menstruation* in the initial stage (Quincke, Leo, Knecht, Scheby-Buch, Obermeier, *et al.*[1]). At the beginning of mall-pox, and especially in the initial stage, the menses set in with striking frequency, whether before their time or at the egular period. We cannot help referring to the premature ppearance of the menses, a not infrequent event in other infecious diseases, also to a direct action of the variola process upon he genital functions, the mechanism of which action is unknown. Moreover, the punctual appearance of the menses is so often observed in connection with the initial stage (before the beginning of the eruption), that it is difficult to regard this as something purely accidental. Obermeier found himself inclined to suggest his interpretation: that the period of menstruation was able to modify the duration of incubation, to cut it short during its later ays, or to lengthen it, and thus to exert a decided influence pon the commencement of the initial stage.

It is but recently that attention has been paid to certain hanges of very great interest and practical importance, which he skin sometimes undergoes during the initial stage. They are he so-called *initial* (*prodromal*) *rashes*. These have indeed een observed before, and some mention of them is to be found in medical literature. But they have been, in part, erroneusly described as independent forms of eruption, occurring imultaneously with that of small-pox, and in part have been casually mentioned as insignificant curiosities. That records of these exanthems are wanting in the descriptions of the best of the older authors, may be most readily explained by the circumstance that they occur with very dissimilar frequency in different epidemics. Thus, in 1870–71, there was abundant opportunity for their observation almost everywhere; while in most other epidemics during the present century they have appeared with comparative rarity.[1] From an anatomical standpoint these exanthems may be divided into two forms: *erythematous* and *hemorrhagic.* The first are either diffuse, and cover more or less extensive parts, rarely the whole body ("scarlatiniform exanthem"), or they are macular or "measly." The *hemorrhagic* exanthems are composed of extremely, small punctate, often pinhead-sized, hemorrhages in the epidermis, which are more or less dense, and at times so crowded together that the impression of a diffuse redness is produced. In a large number of cases the two forms are combined as petechiæ upon an erythematous base. Frequently petechial-erythematous and purely erythematous exanthemata are found at the same time upon the various parts of the body of the same individual.

---

[1] *Quincke*, Annalen der Charité, 1855, Heft I. *Leo*, l. c., page 491. *Knecht*, l. c., . 219 and 220. *Scheby-Buch*, l. c., p. 509 and 510. *Obermeier*, l. c., p. 81.

As a favorite seat of the petechial eruptions we may mention the lower abdominal region, together with the genitals and inner surfaces of the thighs. When the patient lies with his legs in contact, the exanthem presents the form of a triangle, the apex of which is towards the mons pubis, while its base crosses the abdomen transversely in the neighborhood of the umbilicus (crural triangle of Simon). Very often the eruption also extends along the lateral surfaces of the trunk up to the axillæ, and from this point it frequently invades the axillary folds, the contiguous portions of the arm, and the pectoralis major muscle. In the latter case Simon speaks of a "brachial triangle."

The erythematous eruptions, both of macular and diffuse form, do not evince such marked preference for particular parts. They sometimes spread over the greater part of the body. When more localized, it is usually upon the extensor surfaces of the extremities, especially at the joints, and (according to Simon) with especial preference in the form of a streak starting from the ankle, and covering the skin over the *extensor hallicis longus* muscle. In women I have very often seen a macular initial erythema around the nipples, even in those cases in which no exanthem was discoverable upon the remainder of the body.

---

[1] Beyond a doubt, the greatest credit for investigation of the initial exanthemata belongs to *Th. Simon*, whose articles on the subject, cited in our introduction, we especially recommend for closer study.

The time of the appearance of these interesting eruptions, which imprint something extremely characteristic upon the often indistinct form of the initial stage, is tolerably variable. In general, however, the second day may be designated as that upon which they most frequently make their appearance. A later or earlier advent, however, is not infrequently observed; and, indeed, in certain cases they have been seen to appear as the first morbid symptom, before the initial chill, at which time they were, of course, of especial diagnostic importance.

The duration of the initial eruptions is also extremely variable. The erythematous do not last as long as the petechial, usually twelve to twenty-four hours; though cases where they have lasted several days are not as exceptional.

For a long time after the petechial [1] exanthemata have disappeared, and often during the entire duration of the disease, their marks remain in the form of very small brownish or bluish-green spots. We will return, later, to the opinion adopted by Trousseau and Hebra, that these parts remain exempt from the actual small-pox eruption.

The frequency of the initial exanthemata is exceedingly variable. While they appear almost everywhere in certain epidemics, as, for instance, in that of 1870–71, they are rare in other epidemics. In this way only is it to be explained that the keenest of early observers make no mention of them. Were they more constant, their diagnostic, and perhaps likewise their prognostic value would be very much increased. These two points, which in any case are not to be undervalued, will be referred to again at the appropriate place.

---

[1] I intentionally use the term "petechial," as applied to these exanthemata, and endeavor to avoid the expression "hemorrhagic," because the latter might readily create the impression of a certain malignity of this eruption, which belongs to it only in a limited measure, and not at all in the sense in which it applies to actual hemorrhagic small-pox.

The group of symptoms thus far described, as well as the special symptoms of the initial stage, may be greatly modified in certain cases. Many of the symptoms described may be entirely absent, while others may appear with special severity. The intensity of the fever may exhibit the greatest variation, from the slightest grade up to a severity such as is attained in few diseases. The initial stage is thus presented under the most varying phases. We will here recall to mind the fact, that neither in the intensity nor in the mode of appearance of the initial symptoms can any indications be found capable of separating variola from varioloid; and that the severity of the symptoms never justifies the certain expectation of variola and the exclusion of varioloid.

The initial stage not only presents itself under the most variable guise, but it is apt to terminate in a variable manner. Even the mode of transition into the eruptive stage is tolerably variable, as we shall see, according to individual idiosyncrasies, and especially to the intensity of the case. It does not always progress to the characteristic eruption, but the disease may terminate with the initial stage, even before distinct efflorescence becomes apparent. And herein the two extremes of small-pox meet: the *Febr. variolosa sine exanthemate*, the most benignant form, and the so-called *purpura variolosa*, the most malignant form, leading to certain death.

The older authors, especially Sydenham,[1] as well as de Haën, Peter Frank, *et al.*, describe the *Febr. variolosa sine exanthemate*, and at the present time also cases are encountered in every epidemic which admit of no other designation. The symptoms are altogether those of the initial stage. An introductory chill, single or repeated, is succeeded by a fever of variable intensity, often quite severe, with prostration, pain in the head and back, gastric symptoms, and sometimes anginose difficulty. Then, after three to four, rarely six days' duration of the disease, instead of the expected small-pox eruption, recovery takes place, with rapid decrease of temperature. Sometimes one or the other form of the initial exanthemata appears, of which the petechial eruption in the crural triangle is of especial diagnostic value. If such characteristic changes in the skin are not manifest, the diagnosis of *variola sine exanthemate*, in an isolated case, is doubtful; and it is often better, when no weighty circumstantial evidence is present, to leave it entirely *in suspense.*[1] To deny the existence of this form, however, as some do, is certainly going too far; and is only reconcilable with the artless view that the cutaneous affection substantially represents the small-pox process.

---

[1] Sydenham called this form "*Febris variolosa.*"

The *purpura variolosa represents the worst course which small-pox can take in the initial stage.* It leads steadily and rapidly towards death. Under this title we understand those cases in which the process designated "hemorrhagic diathesis," the nature of which is yet unknown, and which, as we shall see, may also appear during the exanthematic stage, imprints its frightful stamp upon the disease, even in its initial stage, or at the end of it, before even the first rudiment of a small-pox pustule can be detected. If this view is firmly held, that purpura variolosa is the initial stage of variola that has become hemorrhagic, the manifold objections disappear which have been made concerning its relation to other hemorrhagic and non-hemorrhagic forms of the small-pox process. It disposes particularly of the doubt, recently revived, whether this form should not be withdrawn from the chain of small-pox processes and assigned to a separate position, inasmuch as the anatomical conditions, especially of the internal organs, indicate results different from those observed in the remaining forms. The anatomical variations present are explained most readily upon the ground that the hemorrhagic diathesis causes death in another stage, in which a different condition of the organs occurs. The uninterrupted transition of purpura variolosa into the hemorrhagic pustular forms is exhibited in those cases in which, in addition to the purpura, there are a few actual (hemorrhagic) papules or pustules.

---

[1] We will here recall the oft-mentioned though rare cases of Febr. variol. sine exanth. in pregnancy, where at times the birth of a child covered with variola has revealed the nature of the previously doubtful maternal disease. Comp. page 9.

Young and robust persons are attacked with striking frequency ; a circumstance for which we are unable to offer a plausible explanation. The best observers are in accord as to the fact, and my own experience leads me to agree with them. I have also seen many invalids and debilitated persons fall victims to this terrible form of the disease; and drunkards, pregnant women, or lying-in women succumb most readily among those previously healthy. The initial stage in these cases usually begins in the ordinary manner with rigor, headache, very intense pain in the back, and great prostration. Very soon (even in from eighteen to thirty-six hours) a diffuse scarlatiniform, rarely macular, redness invades the trunk and extremities, but leaving the face almost always exempt. This redness disappears upon pressure with the finger, and is scarcely distinguishable from an ordinary initial exanthem, and is sometimes suspicious only by its peculiar intensity. In this erythema, petechiæ and larger cutaneous hemorrhages rapidly appear, and vary in size up to that of a silver half-dollar. They are usually small and discrete upon the extremities, and confluent on the breast and abdomen in large irregular figures. The face is rendered red and puffy, the conjunctivæ bloodshot, and large black rings are formed around the eyes, through hemorrhage into the cellular tissue of the lids and their contiguous parts. The countenance thus presents a truly frightful appearance, and the patient is often wholly unrecognizable. The tongue is usually thick, with a whitish-yellow coat, and looks almost as it might do if it had been boiled. Diphtheritic processes take place in the pharynx, and a horrible *fœtor oris* increases the loathsomeness of the entire symptoms. There is almost always severe pain in the præcordial region, frequent nausea and vomiting of bilious and often bloody matters, thin bloody stools, and turbid and often offensive urine. With this there usually is a troublesome cough, with serous and likewise bloody sputa ; and in women, particularly when, as is common, menstruation, abortion, or lying-in coincides, a violent metrorrhagia continuing until death. A suspicious quantity of albumen is almost always present in the urine before the occurrence of hæmaturia.

The temperature in purpuric cases does not usually attain any considerable elevation. In nearly all cases observed by myself I found it but seldom more than 40° C.—104° Fahr.—and that usually just before death. Towards the end, the body, particularly the trunk, has a blackish or leaden-gray hue. The intelligence is unimpaired during the whole course, usually until very shortly before dissolution. Only a few patients are fortunate enough to fall speedily into delirium or coma. Zülzer has frequently observed partial or general hyperæsthesia or anæsthesia of the skin, and even paralysis, especially of the extremities. The course of the entire process is extremely rapid. Some patients die within three days after the beginning of the symptoms, and some even earlier. According to my observations it is exceptional for them to survive the sixth day.

Except in the cases described, where the small-pox process is cut short in one way or another during the initial period, the patient, after the expiration of this stage, enters that in which the characteristic small-pox eruption appears and develops. More in the interest of superficial description than in accordance with nature, this period has been again subdivided into two, gradually merging into one another; that of the development of the eruption, *stadium eruptionis*, and that of its blossoming and maturity, *stadium floritionis* (*s. maturationis, s. suppurationis*); then follows (in cases not previously fatal), the stage of retrogression, the drying of the eruption, and the final fall of the scabs, *stad. exsiccationis et decrustationis.* As in the initial stage, the disease exhibits the greatest variations in this period also, in regard to the manner and intensity of its manifestation, dependent principally upon the grade of the cutaneous affection.

We shall see our way most clearly in the multiplicity of these forms if we *first describe the morbid appearances of true variola as a prototype*, and then diverge to the severer forms on the one hand, and the varioloid group on the other.

Regular Course of Variola vera.

Stadium eruptionis.—With a certain regularity, lacking in varioloid, the beginning of the small-pox eruption upon the skin ensues during, or shortly after, the third febrile exacerbation of the initial stage. The eruption almost always appears first upon the face and the hairy scalp. In the latter location, as a matter of course, it is seen only when the hair is thin. It appears in the form of slightly elevated maculæ, pale red at the outset, more or less thickly studded, and varying from a millet seed to a pin-head in size. The forehead, eyes, alæ of the nose, and upper lip are usually covered first. The eruption increases, with a sensation of slight burning and itching, without, however, becoming confluent, even in the severest cases. At the commencement, when the patients are cold or the facial capillaries are contracted from other causes, the traces of the eruption may be temporarily difficult or impossible of detection; but it again appears distinct as soon as the patients become warm.

Somewhat more tardily, usually a few hours later, small red points appear upon the body and extremities; not all at once, in the majority of cases, but in such succession that the back, breast, and arms exhibit the efflorescence at first, the body next, and the legs and feet last. The eruption is almost always less dense upon the trunk than on the face, and remains so. The breaking out of the eruption in other parts of the body at the same time as upon the face, or sooner, is very rare in true variola, though more frequent in varioloid. A minute study of the red points teaches us that they have a certain preference for locating themselves around the hair follicles and the orifices of the sebaceous glands, and often, also, those of the sudoriparous glands; a circumstance which is of importance for the explanation of a manifestation to be subsequently described.

The definite number of efflorescences is usually reached in from one to two days, new points, meanwhile, making their appearance in the spaces between the older ones. Occasionally, however (and here again more frequently in varioloid), fresh points appear still later, also between those further developed. In their normal course these points pass through certain changes, until they eventuate in the complete formation of the pock. On the second day of the eruption, the fifth of the disease,[1] these spots have become more darkly reddened, enlarged in diameter, and elevated into distinct papules. On the sixth day of the disease (third of the eruption), having meanwhile become more conical, they present at their apex a vesicle filled with a clear opaline fluid. This vesicle enlarges until the seventh or eighth day, when it is usually the size of a pea, and nearly hemispherical in form. As they continue to enlarge, a central depression is found in a greater or less number of these vesicles. This is the so-called umbilicus, at the bottom of which the opening of a hair follicle or sweat gland is frequently observed. This circumstance, as will be shown in the anatomical division of our subject, is closely connected with the origin of the umbilicus, concerning the nature of which so much has been said. A large number of distinctly depressed vesicles, which have neither a central opening nor a hair, proves, however, that this is not a *conditio sine quâ non* for the production of the umbilicus.

---

[1] For the sake of computation we must fix the duration of the initial stage at three days, which indeed for variola vera is very near the rule. Of course the periods assigned as the duration of the phases of development of the eruption are likewise not infallible, but only of average value.

If the vesicle be punctured at a circumscribed point, a small yellowish drop of lymph escapes through the opening, by no means its entire contents. This observation leads to the conclusion that the interior is not uninterrupted, and that the fluid is not entirely free. In fact, anatomical examination, as will be more fully discussed hereafter, reveals a sort of cellular construction of the vesicle, of course genetically and morphologically, in a sense somewhat different from that understood by the older authors who occupied themselves a good deal with this point.

Stage of suppuration.—About the ninth day of the disease the contents of the pustules, which have gradually been growing turbid from admixture of pus corpuscles, become completely purulent. The disease then enters the stage of suppuration. Upon complete maturation of the pustule the red edge of its base becomes considerably broader and darker (*halo* of the pustule). The skin in the immediate vicinity undergoes considerable tumefaction at the same time, and this swelling becomes confluent where the pustules are thickly set, greatly increasing the dimensions of the affected parts. The head, in particular, attains a frightful thickness in this manner; the face swells to a shapeless mass, and the patient becomes absolutely unrecognizable. Those portions of the skin of the face where the connective tissue is lax as the eyelids and the lips, become very œdematous, even under the influence of few pustules. The scalp, according as it happens to be the seat of pustules, often undergoes a great amount of swelling, which is so painful that simply lying upon the occiput becomes unendurable, even in the case of patients but little sensitive.

We have already mentioned that the eruption is apt to appear on the trunk and extremities somewhat later than on the face. In like manner the pustules on these parts pass through the metamorphoses described, from one to several days later; but without any further essential difference. Consequently suppuration may be complete on the head while vesiculation is just commencing in the efflorescence on the legs. Even when the eruption on the trunk is perfectly discrete, that on the hands is frequently very dense. This occasions, especially upon the fingers, where the eruption readily becomes confluent, extremely violent pains, in comparison with which all the other grievances often disappear. Next to a greater nerve supply, this very peculiar susceptibility is due to the fact that in the skin of the fingers very contracted sacks are tightly fastened around thin cylinders of bone, which must be subjected to great tension by the slightest tumefaction, inasmuch as yielding is impossible.

When we recall the suffering occasioned by a single paronychia, we may estimate the torture of a small-pox patient, who really has a paronychia on each finger. The feet, and especially the toes, are in conditions quite similar to those of the hands, except that they are less thickly covered with pustules. The character of the efflorescence differs somewhat on the callous portion of the fingers and toes, inasmuch as the unyielding nature of the horny epidermis admits of but little or no projection. The eruption is therefore usually presented in the form of simple, or very slightly elevated, maculæ.

Of the different regions of the trunk the hypogastrium, and especially the crural triangle previously mentioned, are often but sparsely covered or quite free from the eruption, even when it is very abundant elsewhere. Many (Hebra, Trousseau) are inclined to bring this into a causal connection with the initial exanthem, the favorite seat of which is just this locality. But cases may be seen in every epidemic in which no initial eruption has existed, and yet the locality in question has remained comparatively exempt from pustules, so that it may with justice be asked: Have we here really to do with cause and effect, or do not both phenomena exist independently of one another; so that the region in question (from causes as yet unknown) is firstly comparatively protected against the pustules, and secondly, presents a favorable soil for the initial eruptions? Frequently as the circumscribed exemption of the hypogastric region impresses us, when other parts are covered with an abundant eruption, it is not rare, also, to see this region thickly covered with pustules (even after a preceding initial eruption).

Portions of skin, upon which mechanical or chemical irritation has acted, either before infection or during the stage of incubation, are usually affected in a very characteristic manner. Even when the eruption is extremely scanty upon the remainder of the body, the pustules here are usually very abundant, and frequently even confluent.

This behavior of the exanthem comes most frequently under observation where, shortly before, irritating inunctions or pencillings with iodine have been made, sinapisms or drawing plasters applied, or contusions or superficial erosions have taken place. A case especially striking in this regard presented itself to me in a man who had been seriously infested with body-lice before his infection. He came in with a varioloid of moderate severity, and exhibited numerous scratches extending over the whole body, studded with pustules, crowded thickly one upon another like pearls on a string, and partly confluent. Many of those streaks were three to four inches in length, and, at a distance, gave the patient the appearance of having been tattooed.

It would seem easy upon these interesting facts to construct theoretical conclusions respecting the causes of the density and distribution of the variola eruption in ordinary cases, but we do not get beyond the preliminary speculations. Further observations, however, and especially experimental research, may prove of great value in this inquiry, and perhaps clear up the principal points. I will not omit to mention that, according to my experience, the conditions of the skin in question give occasion to this peculiarity of the localization of the pocks only when existing *before infection* or *in the stage of incubation*. On the contrary, when I produced such conditions of the skin experimentally, in the initial stage (by sinapisms, painting with iodine, etc.), the eruption was never thicker here than on other localities.

Almost simultaneously with the appearance of variola upon the external integument, small-pox eruption occurs upon the *mucous membranes* in general, wherever they clothe the orifices of the various canals of the body. This eruption is scarcely ever wanting on the mucous membrane of the mouth, pharynx, and contiguous regions. As the intensity and extent of the eruption is generally very variable in different cases, so the different portions of the mucous membrane are apt to be covered in variable degrees. Sometimes it is the inner surface of the cheeks and lips which is principally affected; sometimes, and this is more frequent, the soft palate and tonsils, as well as the remaining pharyngeal structures, are severely attacked by the eruption; and this condition is often followed by secondary phlegmonous inflammation, with the formation of abscesses in the tonsils and arches of the palate. Thence the eruption not infrequently extends farther upon the mucous membrane of the pharynx, the larynx, and trachea (decreasing in density as it extends in depth). Hoarseness is then associated with the dysphagia, and sometimes even complete aphonia. Deep ulcers are formed not infrequently in the laryngeal mucous membrane, and even perichondritis, with secondary necrosis of the cartilage, and sometimes acute œdema of the glottis; from the mucous membrane of the trachea the process sometimes extends into the large bronchi. The tongue, which always appears heavily coated, is, in some cases, more or less thickly studded with pustules. Often, too, it is so strikingly free from them that, in its normal size and smoothness, it contrasts characteristically with the greatly swollen mucous membrane of the rest of the mouth. Cases, fortunately rare, occur, in which the parenchyma of the tongue becomes secondarily phlegmonous, and the organ is enormously enlarged, so that often it can no longer be retained wholly within the mouth (*Glossitis variolosa*). The older authors, under the influence of ideas based upon the humoral pathology, busied themselves a great deal with the salivation not infrequently existing in cases affecting the mucous membrane of the mouth, and which may be so copious that the secretion runs continuously from the mouth. We now either recognize its origin in parotitis, or regard it simply as a reflex symptom induced by the inflammatory irritation of the mucous membrane of the mouth. From the pharynx, the small-pox process usually extends into the nasal cavities, so that these passages, swollen and filled with scabs, sometimes become obstructed, and breathing may be seriously impeded, especially if the tonsils and larynx participate. The eruption has often been observed to extend posteriorly as far as the orifice of the Eustachian tubes (Wendt). The eruption occurs less frequently upon the mucous membrane of the vulva, vagina, and lower portion of the rectum; and then it is later in appearance and comparatively sparse. Annoyances of these parts, therefore, are often first instituted at the very time when those of the mouth are already on the decline. The urethra is almost always exempt from pustules. At most they are quite isolated, and close to the meatus.

The pocks on the mucous membranes are somewhat different from those on the skin, as can be most readily observed in the mouth and pharynx. They appear first as whitish or pearl-gray elevations upon a reddened base; when present in considerable numbers the mucous membrane is usually diffusely swollen. The efflorescences do not develop to such large and complete vesicles as those on the skin; but, under the influence of the warm and likewise macerating fluids of the mouth, they very soon lose the epithelium at their apices, thus undergoing slight losses of substance. When the pocks are confluent these abrasions usually unite into larger irregular excoriations. As a matter of course, the pain, at first only moderate, is considerably augmented by these ulcerations.

We will now glance at the changes in the *general condition* of the patient during the development and progress of the exanthem.

With reference, in the first place, to the *febrile symptoms*, we have already mentioned that the first signs of the eruption usually appear upon the skin, at the height of the third febrile exacerbation. In mild forms of the process the temperature falls rapidly, and often declines to the normal degree at once, or falls still lower. In variola vera it often remains for twelve to eighteen hours, according to the intensity of the case, at about the highest degree reached, or rises at evening even a tenth higher. Then the temperature undergoes a similar but much slighter and more gradual decrease, during which transition the normal temperature is reached; not altogether in severe cases, but quite so in rather mild ones. The descent of the temperature occurs either in a remittent type, often with considerable evening exacerbations (in somewhat milder cases), continuously, the evening temperature being but slightly higher than that of the morning, and sometimes the same, or even less.[1] The pulse, in uncomplicated cases, corresponds, as in most of the stages of the disease, with the temperature, varying between 90 and 112 to 120 beats in the minute.

The remaining troubles of the patient usually become considerably lessened when the eruption commences. There may be almost absolute comfort, the remission usually bearing a direct proportion to the remission of the fever. The pain in the back, the vomiting, and the tormenting headache moderate or cease entirely; the delirium abates, and a salutary rest, or even sleep, ensues. Such absolute comfort as is experienced at this time in varioloid occurs very rarely in variola vera.

As soon as the contents of the pustules begin to be purulent, the febrile condition, which had been greatly lessened during the eruption, again augments in a notable manner (*Febr. secundaria s. suppurativa*). Sometimes this *suppurative fever* (most frequently in the case of very sensitive persons, women and children) is ushered in by a chill, or by repeated chills, and continues from three to six, or even eight days, according to the severity of the case. When moderate, the temperature at evening rises to about 39-39.5° C. [= 102-103° F.], and (in the absence of complications) seldom rises above 40° C. [= 104° F.], and then only in the severest critical cases.

The form of the curve in this stage is very different from that in the initial fever. It has chiefly the remittent type, and daily variations of nearly 2° F. are not rare. The pulse fluctuates between 100 and 120 beats in the minute. The course and height of the suppurative fever are, in uncomplicated cases, directly dependent upon the cutaneous affection, its intensity also being greater the severer the dermatitis. With the suppuration the other sufferings of the patient, who rejoiced too soon in the eruptive stage, again undergo a marked increase. With considerable augmentation of the pain, and an increasing sensation of heat, great disquietude and absolute sleeplessness usually again occur. The headache returns, and the disturbances of the sensorium, especially delirium, form a complication as fatal as it is frequent.

---

[1] In severe cases the temperature during its descent does not fall below 100° F. in the morning, nor below 102 F. in the evening.

This mental disturbance often attains such decided severity that it sometimes becomes dangerous both for the patient[1] and those in his vicinity, and the most careful watching is imperatively requisite, and even the use of the strait-jacket where attendants are deficient. The delirium is partly occasioned by the intensity of the fever, but is partly, also, as it seems to me, dependent on hyperæmia of the brain, associated with the often colossal tumefaction of the skin of the head and face. Alcohol, too, often plays an important rôle in the causation of delirium; and unmistakable delirium tremens is well known to belong to the most frequent complications of variola. The delirium sometimes continues day and night, and when the patients are conscious in the daytime, sufferings of the most varied kind, burning pains in the face and in the hands and feet, affections of the mucous membrane, etc., keep them in continuous disquietude and excitement.

About the eleventh or twelfth day—sometimes later, seldom earlier—the *drying of the pustules—stadium exsiccationis*—begins, and with it an alleviation of the numerous severe local and general symptoms. The desiccation naturally commences in those parts in which the eruption first appeared, and therefore usually on the face. Even before the eleventh day, and sometimes as early as the eighth or ninth day, an exudation of a viscous fluid occurs upon the surface of the pustules; at first yellowish and honey-like, and forming, upon further desiccation, a firm, somewhat rough coating. The remainder of the contents of the pustules now dry speedily also, and then small brownish scabs are formed, at first rather firmly adherent to the surface. With the continued desiccation of the pustules, the redness, swelling, and tenderness of the skin lessen, the eyes again open, the nasal passages become patulous, and the countenance resumes its proper appearance.

---

[1] I know of a case where such a patient, in his febrile fury, stabbed himself.

On the trunk and extremities, where desiccation begins somewhat later, the pustules become ruptured in many localities, their purulent contents, soaking into the bed and body linen, undergo decomposition upon the skin and in the clothing, and thus a highly offensive odor becomes disseminated. Many pustules dry into brown scabs without rupturing. These are usually those small ones which are observed upon certain parts of the body, *e.g.*, the extremities, or in certain cases over almost the whole body. On the palms of the hands and soles of the feet the drying usually commences earlier than on the remaining portions of the extremities, although the scabs adhere longer. This circumstance, as well as their peculiar, flat, lenticular form, is dependent upon the greater thickness of the epidermis, which is here quite horny in the laboring classes. Just as we saw the efflorescence, in the suppurative stage, according to the compactness of the epidermis, form only flat prominences, or none at all, so, after drying, the scab, which retains the flat lenticular form of the pustule, remains for a long time encapsuled between two thick layers of epidermis, and is often artificially extracted by the intolerant patient from beneath the horny layer.

An often irresistible *itching* takes the place of the pain at the period of desiccation, so that many patients can only with difficulty refrain from scratching. The general belief, that scratching renders the resulting scabs less sightly, is unfounded, except for such lesions as may possibly be directly due to the scratching. After the complete formation of the scabs, at a time varying in accordance with individual conditions of the skin, and the grade of the disease, they fall off gradually, and likewise with variable rapidity.

After the fall of the scabs, *pigmented* and often *slightly elevated maculæ* almost always remain, which sometimes become pale and sometimes extremely hyperæmic, under the influence of changing temperatures. The whole body of the patient appears spotted, and this, unfortunately, is particularly marked in the face, so that the individuals often present a truly frightful appearance, and are hardly in a condition to appear in public. According as the pocks (as will be more minutely discussed in the anatomical division) have been limited in their seat to the epidermis, or, as in severe forms, have involved and destroyed the papillary portion, these maculæ, after the pigmentation has subsided, may disappear without any traces whatever, or else *depressed radiated scars* remain, which in time become whiter than the surrounding skin. On narrow folds of skin, especially on the nostrils, and on the nose in general, this loss of substance takes place in a particularly unsightly manner; so that the borders of the alæ of the nostrils often appear indented, and the ridge of the nose and its tip appear split and torn.

As already remarked, a considerable improvement takes place in the general condition of the patient with the commencement of decrustation; and this continues, in normal cases, until recovery is complete. The fever in particular undergoes a decrease more rapidly or gradually according to the grade of the dermatitis; so that the patient is completely free from fever in a short time. It must be asserted here, in opposition to the views of many, that a subsequent rise of temperature is not occasioned by the normal course of desiccation. Where this does occur, and it is no rarity in severe cases, it is referable to complications, especially to erysipelas, phlegmonous processes, and more or less extensive furunculosis; while internal affections are rare at this period.

When the patients are not affected in this manner they become quiet, sleep well, have a strong desire for food, and only suffer slightly from the constipation which has existed throughout the entire disease. With the fall of the scabs, often also somewhat later, many patients lose their hair, especially if the eruption has been abundant upon the scalp. The prospect of its return is favorable where the pocks have not been very deep; while, of course, this is not the case if the process has involved and destroyed the hair follicles to any great extent. With many persons it is only necessary to grasp the hair, and barely pull it, in order to remove a handful of loosened locks. Loss of the nails is much more rare, though more frequent in the confluent form about to be described.

After regular uncomplicated variola vera, with a moderate course, five or six weeks usually elapse before convalescence is complete, while severe cases often last much longer.

We have now presented variola vera in the first place as a prototype, solely, as was remarked, for the sake of superficial description. The remaining forms of the small-pox process may present certain complications, according to the general condition of the patient, and especially the intensity of the skin affection. These variations from the course described may present the most extreme limits from the severest and absolutely fatal forms to the very lightest cases of varioloid, in which but a few small pustules reveal the fact that we are dealing with a sick patient. *None of these forms are sharply defined amidst the great group of variolous affections; but there is rather a gradual transition from one into the other, so that general outlines* are to be associated with the most customary designations *rather than sharply circumscribed features.* According to the individual tendency of different authors, more or less numerous special forms are described in medical literature, and designated with special names, in part arbitrarily based upon subordinate peculiarities. We are neither able nor desirous of considering all these, and will call attention at once to a particularly severe form of small-pox, the so-called

Variola confluens.

Even the initial stage in this variety runs an unusually severe course; at all events, mild symptoms at this time justify the exclusion of confluent small-pox with tolerable certainty. The initial fever is unusually violent, and the eruption in many cases develops far more rapidly than in ordinary cases; frequently twelve, or even eighteen hours earlier. The spread of the eruption over the whole body is usually completed more quickly too than in the other forms, and often does not require more than thirty-six hours. Indeed cases are not infrequent in which the eruption is completed almost simultaneously on the face and on the other portions of the body. The spots are particularly numerous on the face and hands, and the maculæ are in such close juxtaposition on the head, even on the first day of the eruption, that they appear almost confluent. On the second day the skin is already pretty uniformly swollen, and intensely reddened, and so thickly studded with large flat papules, that they rapidly coalesce in large tracts, though individually smaller than those of discrete variola. In the ordinary progress of things suppuration speedily follows, and flattened, irregularly outlined, confluent, yellowish-colored prominences are seen upon a darkly reddened and diffusely swollen skin. With the progress of their development these patches run together over still larger surfaces, the remaining septa disappear, and the epidermis is elevated in the form of large flat bullæ filled with sero-purulent fluid. In this manner the entire skin of the face may be converted into a bulla, and the patient appears as absolutely unrecognizable as though concealed by a mask.[1] While in severe cases the pocks may completely coalesce on the face and hands, on other parts of the body they almost always remain discrete, even though closely crowded together, and at all events never become confluent except over limited surfaces.

The *mucous membranes* in this form are almost always attacked with extreme severity, so that the condition of the patient, which was in other respects deplorable, becomes thereby aggravated to an unbearable degree; indeed, in some cases, this complication is the immediate cause of a fatal termination. Upon the mucous membrane of the mouth and throat the eruption is usually confluent, and even diphtheritic. Affections sometimes ensue which may spread over the soft palate, the tonsils, the posterior wall of the pharynx, and thence into the nasal cavities. The above-mentioned glossitis variolosa, if it occur at all, is most apt to make its appearance in confluent small-pox. The larynx is attacked with marked severity in almost every case, the affection often eventuating in the formation of submucous abscesses, necrosis of the cartilages, and acute œdema of the glottis. While in the other forms of variola the conjunctiva either remains entirely free from the exanthem, or is affected in but a moderate degree, in this variety it is beset with points of efflorescence in great numbers, which unfortunately sometimes lead to purulent forms of keratitis with perforation. The affection in the buccal cavity is often accompanied by inflammation of the parotid glands, probably depending upon an extension of the inflammatory process along Steno's duct. A very annoying symptom also is a severe salivation, which is frequently aggravated on account of the violent pharyngitis which renders deglutition almost impossible.

---

[1] The comparison is frequently made—and it certainly is true of a few cases—that the face looks as if it were covered with a parchment mask, or sprinkled with sand.

The gravity of the *derangement of the general condition* corresponds to the severity of the local manifestations. Prominent among the symptoms of the general derangement is *fever*, which is here characterized by great severity. The temperature, which, as we have seen, attains an extraordinary height (106°-110° F.) in the initial stage, falls very slowly after the appearance of the eruption, and only very slightly. It remains until suppuration at 103°-104° F., and then again rises even higher than before. Violent delirium is of very common occurrence in confluent small-pox, and not infrequently coma takes place. Furthermore, a vast number of complications are peculiar to this variety of the disease: inflammations of the serous membranes—especially pleurisy and pericarditis—pneumonia, both croupous and lobular, the latter ensuing upon the violent and obstinate bronchitis which but few patients who suffer from variola confluens escape. Many persons are tormented by *violent, uncontrollable vomiting and retching*, and in some epidemics, according to older authors, especially obstinate diarrhœas are observed, which, beginning in the initial stage, last throughout the entire disease. In the majority of confluent cases a considerable degree of albuminuria is present, which is fully accounted for by the changes which take place in the renal parenchyma, and which will be described under the division of pathological anatomy.

The integument often presents, besides the variolous eruption, certain other severe affections, the most prominent of which are *multiple abscesses, extensive phlegmon and erysipelas, and even gangrene* in those places where the confluence is most pronounced.

The *mortality* in confluent variola is of course enormous. The patient may succumb at any stage of the disease. Frequently they pass through the entire torment and suffering preceding the period of desiccation, and finally die in this stage from exhaustion or from the complications. More rarely death is preceded by marked symptoms of pyæmia, which occur now and then during every extensive epidemic. In still other cases deaths take place at an earlier period in the course of the disease, sometimes even in the stage of eruption, following adynamic symptoms with an extremely high temperature (107°-109° F.). The older pathologists regarded the latter as a special form, under the name of "*variola typhosa seu adynamica.*" It is hardly possible to conjecture under just what circumstances small-pox assumes a typhoid character. This condition may, however, ensue in the discrete form of the disease also.

If confluent small-pox terminates in *recovery*, the convalescence takes place very slowly, and is usually interrupted by arious sequelæ, among which the above-mentioned cutaneous omplications (particularly furunculosis) play an important part.

In the stage of *desiccation* large coherent scabs are formed in he confluent patches, which for a long time remain firmly adhernt to the skin, while beneath them suppuration of the papilary layer, which is always markedly implicated, generally coninues for some time. After the scabs have fallen off, deep osses of substance are left behind in the cutis, giving rise to xtremely ugly scars which, in the face especially, often proluce permanent and very unsightly disfigurements. Permanent lopecia is also a not infrequent sequel of confluent small-pox.

Variola Hæmorrhagica Pustulosa.

We have already seen that, in consequence of hemorrhagic liathesis, the small-pox process may prove fatal even in the nitial stage. But aside from this form, which is known as *purpura variolosa*, *hemorrhagic symptoms may also occur at lmost any time in the stage of efflorescence*, and in order to listinguish this group of symptoms from *purpura variolosa* he term *variola hemorrhagica pustulosa* may be employed. It vill be at once seen that this nomenclature is rather for the sake f convenience than designed to express any essential difference between the two affections. The cases observed in every epidemic where extensive hemorrhages of the skin and internal organs occur, in connection with a few sanguinolent abortive pustules, form a connecting link between the two forms.

The characteristic hemorrhages into the pustules in the hemorrhagic form of variola take place under various circumstances. It occasionally happens that the papules become hemorrhagic immediately after they are formed, or even at the moment that they are developed; in other cases the papules first become vesicles, and the change then occurs in the contents of the latter, and in still other cases the hemorrhage first takes place in the pustules, after they have attained nearly their ultimate form and size. Again, a great diversity is manifested in that the eruption sometimes becomes hemorrhagic over the entire body, with petechiæ and ecchymoses appearing between the spots of efflorescence, or the larger portion only of the eruption may be so affected, or the half only, or even but a small portion of it. The interesting modification where extensive hemorrhages occur in the skin during the initial stage, and the patient lives long enough for simple (non-sanguinolent) pustules to develop between the hemorrhagic spots (Reder, Hebra's Handbuch), I am unable to vouch for from personal observation, nor, as it appears to me, can the majority of recent observers.

The most frequent of all these forms is that in which the pocks become hemorrhagic after they have attained about the size of a lentil. In these and in the rest of the hemorrhagic cases, the hemorrhage does not occur all at once, but by degrees. I have almost invariably noticed that the hemorrhages begin upon the lower extremities, and on this account I deem it advisable, whenever there is apprehension of this unfortunate event, by all means to examine this region very carefully.[1]

---

[1] In this connection I would suggest the following caution: Cases occur, especially in delirious patients, who leave their beds and run about, where pustules of the lower extremities become filled with blood in a purely mechanical manner (as happens in ulcers of the leg under similar circumstances). That this occurrence is no dangerous symptom, is proven by the normal course of the disease subsequently.

I remember distinctly the case of a waitress, twenty-one years old, who entered the hospital in the suppurative stage of a very moderate attack of discrete variola, which

In connection with the hemorrhagic eruption livid spots of variable extent appear upon those *mucous membranes* which are exposed to the atmosphere, as well as upon the external integument. The mouth and throat, with the parts adjacent, are principally affected. Very often diphtheritic affections, especially of the pharynx, velum palati, and tonsils, are present, which infect the neighborhood of the patient with an extremely offensive odor. Moreover, in many cases there is a peculiar sponginess of the gums, which are of a dirty hue and readily bleed, closely resembling what we see in scurvy. Next in frequency come the hemorrhages from the nose, lungs, rectum, kidneys, and uterus, already described in connection with purpura variolosa. In females metrorrhagia is commonly present, being consecutive either to childbirth, abortion, or menstruation. Scarcely less frequent are hæmaturia, conjunctival hemorrhage, and bloody stools, while bloody expectoration and hæmatemesis are somewhat more rare. Sometimes but a single one of these symptoms occurs, and as regards their intensity and persistency cases differ in a very great degree.

The *general condition* is often considerably disturbed from the very beginning. But neither from the character nor intensity of the symptoms is it possible to predict with any certainty the subsequent development of the hemorrhagic form. The constancy and unusual intensity of the pains in the back appear, however, to be somewhat characteristic, as has been mentioned in connection with purpura. When a post-mortem examination reveals extensive hemorrhage in the renal pelves and in the renal and retroperitoneal tissues, it is very natural to refer these pains to the hyperæmia of these parts which has obviously existed. Frequently the initial stage is marked by an exceedingly violent fever, while in the eruptive period, and, in fact, during the entire subsequent course, the temperature remains comparatively low. Cases are seen, especially in aged persons, or those debilitated

---

was running a perfectly normal course, on whose feet and legs, up as far as the thighs, nearly all the pocks appeared to be filled with blood. Under the influence of a slight delirium potatorum, and being poorly attended, she had been rambling about, out of bed, as we were informed, almost the whole of the day and previous night. The accompanying card of admission exhibited the ominous diagnosis, "variola nigra."

from other causes, in which the bodily temperature during the entire course scarcely runs above 102° F., or even fails to reach that. When the fever is very intense at the beginning, it often remains of a high grade until death occurs. The curve of temperature in variola hæmorrhagica bears scarcely any resemblance to the typical curve of the common variola vera. The pulse very early exhibits a considerable frequency, which is in contrast with the relatively low temperature of the body. Where extensive hemorrhages have taken place from internal organs, and continue until death, we often observe a rapid fall of the temperature (to 81° F., or even less) just before the fatal termination, while, at the same time, the frequency of the pulse is increased to 140–160.

The *course* pursued by the hemorrhagic small-pox is usually more protracted than that of purpura variolosa, but almost always quite as fatal. The cases which terminate in recovery are extremely rare, and where one does occur, only a few pustules, or at least not all, become hemorrhagic, and these usually at a relatively late period. The absence or slight degree of hemorrhage in the internal organs may also be a favorable sign. Should a patient have the rare fortune to survive, the convalescence is very slow, and for a long time accompanied with evidences of extreme inanition.

The special *etiology* of this form of variola hæmorrhagica is quite as obscure as that of purpura variolosa. Somewhat in contrast with the latter, the pustular form occurs more frequently in older persons, usually in those over forty years of age. Weak, sickly persons and convalescents, as well as pregnant women and those in childbed, appear also to be particularly predisposed to this form.

Varioloid.

From the severe forms of small-pox which we have thus far considered, let us now turn to the varying phases of its mild course, which are included under the name of Varioloid. (Variola modificata seu mitigata). The opinion that varioloid is a special disease, differing in its nature from variola vera, has long since been given up. It is generally conceded at the present time *that varioloid is nothing more than a form of small-pox with a milder course and a shorter duration*, and this view being accepted it is readily seen that between varioloid and variola vera no absolute line of distinction can be drawn. During every epidemic of any considerable extent a number of cases are found which show a transition from one to the other form, and which, even when we have followed their entire course, leave us in doubt as to whether we shall call them cases of "variola" or "varioloid." The quantity or quality of the eruption is as far from being a good criterion in determining the nature of the affection as the presence or absence of the suppurative fever, which some consider as decisive in this respect. The latter does not even depend entirely upon the intensity of the disease, but upon personal peculiarities, and particularly upon the sensibility of the person attacked. As regards the peculiar conditions under which varioloid occurs, it is important in the first place to observe *that many persons are only attacked by this form, on account of a naturally slight susceptibility to the small-pox contagion.* We find, accordingly, frequent mention made of cases in which the disease runs an extremely mild course. A circumstance which has a still greater influence upon the occurrence of varioloid lies in the fact of the individual having had a previous *attack of variola*, or in recent times this influence is generally due to *vaccination.* One attack of true variola, or likewise of vaccinia, protects most persons completely from the action of the small-pox poison for a certain though variable length of time, but the longer the period that has elapsed since the termination of the protective process, the less will the influence of the latter be. Thus individuals, whose immunity from the disease has in this way become impaired, are capable of withstanding the contagion of small-pox to only a limited extent, and consequently undergo a modified form of the disease, namely, varioloid. At the present time vaccination exceeds by far all other agencies in the production of varioloid, *and under its influence this form has become very much more frequent than in former times.*

A prominent characteristic of varioloid is, that it exhibits *far greater variations and "irregularities" than variola vera, in respect to the duration and course of its stages and the character of its symptoms.* This is manifest even in the *initial stage.* The widespread opinion that this is always milder than the initial stage of the variola vera, when asserted in this positive way, is decidedly incorrect. To be sure, in very many cases it is mild, and at times almost devoid of symptoms; but, on the other hand, the most violent initial symptoms, with intense fever and marked disturbance of the general condition, are observed by no means infrequently in quite insignificant cases of varioloid. *The length of the initial stage* appears often to be less than in variola vera, lasting sometimes only two days, or even but one. But more frequently a decidedly protracted duration is observed, and in this respect a certain lack of uniformity is evinced, in contrast with variola vera, in which this stage, as we have already seen, lasts three days with scarcely an exception. When the *initial fever* is present, the curve of temperature undergoes a sudden and very rapid descent either just at the outbreak of, or more frequently after, the appearance of the eruption. Thus by the end of the first, or at the commencement of the second day, the bodily temperature has reached the normal degree, or even gone below it. From this time the temperature usually remains normal, and only occasionally at the commencement of suppuration it again undergoes a slight elevation, which, however, seldom lasts more than twenty-four hours. This elevation of temperature is not so much the result of the intensity of the disease as it is due to individual irritability, especially in the case of women and children, and other delicate persons. During the period of desiccation I have never observed febrile excitement unless complications were present.

The *initial exanthemata* which were mentioned above have been designated by many of the older authors (Trousseau) as a group of symptoms belonging to varioloid; but so general an assertion is incorrect. It comes nearer the truth if we except the petechial eruptions, whose favorite seat, as already mentioned, is in Scarpa's triangle. As regards the non-hemorrhagic, purely erythematous eruptions, both macular and diffuse, it may be stated that they precede varioloid almost exclusively. I do not remember of having observed, out of a large number of cases, a single one where severe variola vera developed after a well-marked simple initial erythema. *It appears to me, in fact, that, as a rule, the development of the pocks is less, the more extensive the initial eruption is.* Viewed in this light the purely erythematous initial exanthemata prove to be of decided prognostic value. Where they are well developed we may predict, in spite of the severe disturbance of the general system, that with great probability the form of the disease will be mild, while, after well-marked petechial exanthemata, variola vera will nearly always ensue, and not infrequently the confluent variety.

The true *varioloid eruption* presents a comparatively great diversity with regard to its mode of beginning, form, arrangement, and distribution. In contrast with variola vera, which is so regular in this respect, the eruption does not always begin in the face, but often upon the trunk, or simultaneously on various parts of the body. The length of time from the beginning to the completion of the eruption is also very variable. Sometimes all the pocks appear simultaneously, or in such rapid succession that their definitive number is reached far earlier than in variola vera. At other times late consecutive crops appear, so that, together with far-advanced pustules, we may have incipient spots, papules, and vesicles, and occasionally on a surface of small extent all the various stages of development of the eruption may be seen side by side. Moreover, the form and degree of perfection which the pocks finally attain often differ upon the same individual.

The structure of the varioloid pustules does not vary essentially from that of the other forms. Frequently the pocks develop quite as perfectly as those of variola vera, even to the implication of the papillary layer, together with the formation, ultimately, of distinct and deep scars, so that then the only marked point of difference between them and the pustules of variola vera lies in their smaller number. But more commonly the eruption either does not pass through all the stages, but recedes at an early period (abortive pocks), or else it passes rapidly and imperfectly through the several phases of development, resulting in the production of more or less dwarfed forms.

The abortive retrogression of the points of efflorescence may occur at various periods. Aside from those cases where the entire disease terminates at the outbreak of the initial exanthem, there are also other, though rare, instances where merely papules or vesicles develop, and which are soon followed by desiccation.

When the pustules of varioloid run through all the stages, the ordinary small red spots develop into conical, somewhat acuminate papules, at the summit of which little vesicles with clear fluid contents appear, often within twelve hours (reckoning from the beginning of the eruption). The vesicles rapidly increase in size, till they are as large as a lentil. Sometimes they are umbilicated, and sometimes not, according to their location and arrangement. Towards the end of the third, or by the fourth day, their contents are often slightly purulent, and a red areola makes its appearance, usually without any noticeable swelling of the surrounding skin. Frequently the contents of the vesicles do not become wholly purulent, but remain rather of a sero-purulent character. Such pocks are usually of small size, often not exceeding that of a pin-head or a lentil. They exhibit a broad red areola, ordinarily of an elliptical form, whose long axis usually corresponds with the cleavage lines of that portion of the skin.

From the fifth to the seventh day (of the eruption), and even earlier in less-developed forms, *desiccation* begins. This often proceeds irregularly as regards its occurrence in different regions of the body, corresponding to the irregularity in the appearance of the eruption. The majority of the pustules merely dry up without previously bursting, forming brownish crusts, which are thinner and smaller than those of variola vera; inasmuch as the skin is not very deeply involved, the scabs fall off sooner than in true variola, sometimes leaving slightly pigmented hyperæmic spots, which, however, soon entirely disappear.

Variations in the degree of development of the points of efflorescence are often exhibited upon the same individual, and where this is the case the pocks on the face are usually much more perfectly developed than on the remaining parts of the body. Likewise with regard to *the number of the pocks* different cases present extreme variations, sometimes a single pustule or but a very few appearing, with almost no disturbance of the general condition (Variolois localis), and again a tolerably copious eruption may occur (especially on the face), accompanied by a mild suppurative fever.

The great variability manifested by varioloid has led to the description and special denomination of a vast number of forms, for which the older authors were especially famous. We find, for example, a variol. acuminata, globulosa, crystallina, lymphatica, fimbriata, verrucosa, siliquosa, miliaris, pemphigosa, etc., while in the majority of instances there was no occasion for any special nomenclature.[1] We will only allude to a few of these terms, which designate certain of the commoner or more interesting modifications.

By the term *variolois verrucosa* we understand that class of cases where the eruption does not develop into large, well-formed pustules, but remains in the form of solid, conical papules, which have a small vesicle at the summit containing fluid. When this has dried, and the scab has fallen off, the solid part of the pock remains for a long time, having the appearance of a warty (verrucous) elevation of the skin. This unsightly, though easily treated form, most frequently occurs on the face.

The form which is characterized as *variolois pemphigosa* is comparatively rare. In this the small pustules develop into large irregular bullæ (without the fanlike meshwork), with sero-purulent contents.

A somewhat commoner variety of the eruption is termed *variolois miliaris*. Here, upon diffusely reddened and somewhat swollen portions of skin (more generally in the initial exanthemata), yellowish vesicles of the size of a millet seed ("miliare") are developed, which progress no further, and finally disappear by simply drying up. This form is more frequently seen in conjunction with completely developed pocks than alone by itself.

Where the retrogression of the pustules occurs in such a manner that the contents are absorbed, leaving empty shells which contain only air, the term *variola siliquosa* is used. This rare form generally occurs upon the trunk and the extremities.[1]

---

[1] *Hebra*, as a matter of curiosity, presents a still greater number of forms, with full reference to his authority for them. Looking at this host of terms, we can scarcely ridicule the Chinese, who have described forty varieties of small-pox.

The *mucous membranes* are also very frequently affected in varioloid, although decidedly less upon an average than in variola. Difficult deglutition, hoarseness, stoppage of the nose, photophobia, and lachrymation are quite common occurrences. At times simple redness and swelling of the membrane is present, without the characteristic papules, but in other cases these may likewise be thickly set. Secondary phlegmonous processes are extremely rare in varioloid, while the diphtheritic changes observed in malignant forms of variola scarcely ever occur in genuine cases.

Complications and Sequelæ.

There is no well-marked dividing line between the complications of small-pox and the local affections belonging to the disease. In describing these complications, therefore, we shall be obliged frequently to repeat previous remarks, and also to omit some things which are described by other writers.

We have already cursorily described the most important complications due to changes in the *skin*. The multiple abscesses, which are often formed during the period of desiccation in confluent small-pox, and in severe cases of the discrete form, are especially painful and protract the convalescence. Extensive phlegmonous processes also, and erysipelas, are not uncommon, but gangrene of the skin is rare, except as an occasional occurrence in the scrotum. In the face, besides brownish spots and cicatrices, acne pustulosa of an obstinate type may continue for a long time. It is produced by narrowing or occlusion of the ducts of the sebaceous glands.

The *nervous system* and the *organs of special sense* are especially prone to be the seat of complications. We do not include here the temporary delirium common to most severe cases, but rather those more permanent psychical disturbances which continue after the disease has run its course. Anatomical lesions of the brain are not common, although meningitis and acute œdema sometimes occur. Sometimes we observe symptoms pointing to the existence of circumscribed encephalitis, such as aphasia, of which I have myself seen two cases. Paralysis of the extremities and of the bladder, from complications existing in the spinal cord, have been frequently observed. Westphal has studied these cases carefully, and shown that the symptoms are often due to numerous circumscribed foci of inflammation in the gray and white matter of the cord (myelitis disseminata). Whether epilepsy ever occurs as a real sequela of small-pox, I think very doubtful.

---

[1] In this connection I would mention a form of variola vera which is sometimes described and said to be very malignant. It is called *Variola emphysematica*, for the reason that from the very outset air instead of pus is contained in the vesicles. This is said to be due to septic influences and almost without exception terminates fatally. I am not acquainted with this form from personal experience.

In the *eyes* we see, in most severe cases, conjunctivitis. This is often made more severe by the œdema of the lids, the inability to open them, and the consequent retention of the secretions. Pustules may be formed on the palpebral conjunctiva, and more rarely on the ocular conjunctiva and cornea. Some observers, as Gregory, deny their occurrence in the latter situations. If the cornea does become diseased, perforation, iritis, and suppuration of the globe may follow. In hemorrhagic small-pox hemorrhages in the retina sometimes occur and produce sudden blindness. It is worthy of remark that Hebra, in 5,000 cases of small-pox, only saw eye complications in one per cent.

We have already noticed that the *hearing* often suffers. Chronic suppurative otitis, and caries of the bones, with partial or complete deafness, are not infrequent.

In severe cases ulceration of the *mucous membrane of the nose*, followed by adhesions, may take place, but it is a rare occurrence.

The *joints* may become diseased. Painful swellings, effusions of serum and pus, inflammation of the cartilages and of the bone itself may occur.

Bronchitis accompanies almost all cases of small-pox, and can hardly be called a complication. In some cases, however, it leads to catarrhal pneumonia and pulmonary phthisis. Croupous pneumonia is not infrequent, but death from sudden œdema of the lungs is rare. Pleurisy and pericarditis are tolerably frequent complications. I have seen ulcerative endocarditis in one case of confluent small-pox.

Diphtheritic inflammation of the soft palate and pharynx is said to be a frequent complication; but I believe that confluent pustules with irregular ulceration are often mistaken for it.

Severe *inflammation of the larynx*, ulcers of its mucous membrane, perichondritis, and necrosis of the cartilages produce chronic hoarseness, or even complete aphonia. We have already mentioned that death may be caused by acute œdema of the glottis.

Inflammation of the salivary glands seems to have been more frequent in earlier epidemics than in those of later years. The older authors considered it to be an unfavorable symptom. I have seen it but seldom, and then it seemed to be of little moment.

Complications from changes in the abdominal viscera are not frequent. I have never seen peritonitis, unless produced by a local cause. Sometimes there is persistent diarrhœa, which may continue after the disease has run its course. Sydenham speaks of a variola dysenterica.

After severe cases of small-pox the patient is left much exhausted and weakened for a long time. In such cases I have seen slight œdema entirely due to the anæmia. In other cases dropsy may be due to chronic nephritis, but this is in my experience a rare sequela of variola.

More has been said concerning *pyæmic symptoms* in the period of suppuration and desiccation than is warranted by facts. I have seen two unmistakable cases of this kind, in which abscesses in the liver and lungs were found at the autopsy.

Anatomy.

Our knowledge of the real nature of small-pox is still unsatisfactory. At the present moment the fashionable parasitic theory is invoked to explain the nature of small-pox, as of many other diseases. Inasmuch as opinions in regard to the etiological significance of inferior organisms in diseases have not yet been thoroughly sifted, we cannot now, even provisionally, accord them an altogether definite value in the case of small-pox.

The present aspect of the question demands, however, an incidental exposition. One of the most valuable papers on this subject is by F. Cohn.[1] He found in the fresh lymph, taken from vaccine and small-pox pustules, small rounded bodies possessed of molecular movement and measuring less than .001 mm. When these rounded bodies were watched for some time, keeping them at a uniform temperature of 35° C., they became segmented and formed small chains and masses. Cohn considered these bodies, which had been already described by Keber,[2] as organisms of the lowest grade. He named them Microsphæra, belonging to the family Schizomyceta, and to the group Bacteriacia. He considers these organisms to be probably the vehicles of contagion, certainly not mere chance products.

Besides Keber, other authors before Cohn, namely, Klebs,[3] Erismann,[4] and Weigert,[5] had seen these same bodies in the contents of the pustules. Zülzer (l. c.) lays particular stress upon the presence of these bodies in the wall and lumen of the blood-vessels. He says that in the "fulminant form" of variola they infiltrate the walls of the small arteries, and may even fill completely the smallest arteries of the skin. He believes that these living thrombi are active causes in the formation of purpuric spots and pustules. The arteries and the tubules of the kidneys are, he says, filled with the same bodies, while in the other viscera they are seldom seen. In the blood also, during the first three days of the disease, he has demonstrated the microspheres (but only in purpura variolosa).

These observations render it undoubted that bacteria are constantly found in variola, but they hardly warrant the conclusion that they are the vehicles of the contagion. Nor do I think that experiments made by inoculation with filtered lymph, as done by Chauveau,[6] much increase the probability of this. There are too many technical difficulties in the way of such experiments. A more convincing method would be to follow the growth and development of these bodies during the successive stages of the disease, and to observe the relationship between their growth and number and the intensity and varieties of the disease.

For the present, therefore, we consider all such observations merely as material which may be of use at some future day to assist in elucidating the problem of the contagion of small-pox.

Until very lately our knowledge of the changes in the skin, and of the development and structure of the pustules, was very uncertain, and it is evident that this depended largely on the tendency of earlier writers to theorize instead of accurately observing.

---

[1] Virch. Arch., Bd. 55, S. 229 ff.

[2] Virch. Arch., Bd. 42.

[3] Handbuch der path. Anat., S. 40.

[4] Sitzungs bericht der K. Accad. der Wissenschaft. Math. naturw. Kl. 1868.

[5] Centralbl. 1871., No. 39.

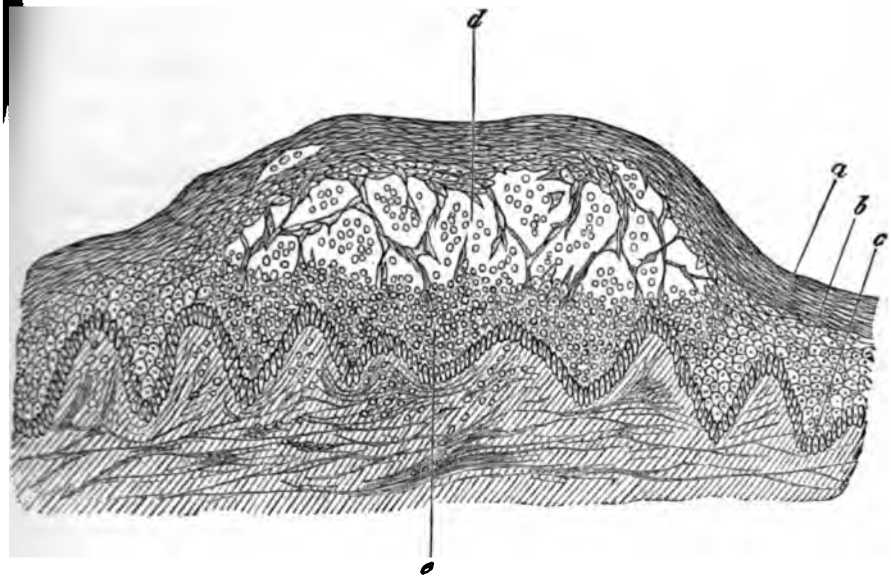
[6] Compt. rend., T. 66, p. 359.

In regard to the *changes in the skin*, and particularly to the *development and growth of pocks* themselves, which is the next matter for our consideration, great obscurity has prevailed until quite recently. The rambling discussions on rather commonplace matters—such as the umbilication and the so-called false membrane of the pock, which have lasted almost to the present time, show that the fault of the earlier investigators was, a propensity to generalize rather than simply to observe.

The study of the different forms of eruption which appear in variola and varioloid shows, as already stated, *that there is no real difference in their structure and development. On the contrary, the different varieties are only due to modifications in their manner and duration of development and of retrogression.* The first trace of the pock, a simple red spot, is produced by a circumscribed hyperæmia of the papillæ, which (according to Bärensprung) is continued through the entire thickness of the cutis. The papule which is formed in place of this red spot is produced by a peculiar change in the epidermis. It differs from other papules, which are due to circumscribed swellings of the cutis.[1] if we examine a papule, we find the cells of the rete Malpighii enlarged and granular, especially the cells situated between the outer epidermis and the layer of cells immediately covering the papillæ. By the swelling of these cells the outer layer of epidermis is pushed up, and flat, solid papules are formed. The next step is an exudation of clear fluid from the papillary layer; this fluid separates the altered cells spoken of above, and lifts up the outer epidermal layer. Thus the papule is converted into a vesicle. The vesicle then becomes larger by the continued exudation of lymph and the swelling of more cells. The exudation does not, however, separate the altered cells from each other. They are separated in small groups, which are compressed by the exudation into membranous and fibrous forms until they look like a network infiltrated with lymph and filling up the cavity of the pock. The epithelial character of this network can be made out in every part, notwithstanding the alteration of the cells by pressure and maceration. This view of the real nature of the network in the pustules is one of the most important advances in the histology of small-pox.[1] While these changes are taking place in the epidermis, the corresponding papillæ become swollen and infiltrated with serum, while their blood-vessels are dilated, tortuous, and surrounded by new cells.

---

[1] It is still undetermined how far the papillæ are changed in the formation of the pustules, whether in all cases or only in the severe ones.



a. Outer layer of epidermis. *b.* Middle layer. c. Cylindrical cells of the rete Malpighii resting immediately upon the papillæ. *d.* Reticulated cavity of the pock, containing pus-corpuscles, with the epithelial framework. *e.* Purulent infiltration of the middle layer of the epidermis.

In many pocks, as has been said before, soon after the formation of the vesicle, and advancing with it, a *central depression* or

---

[1] This view of the structure of the network was first demonstrated by *Auspitz* and *Basch* (Virch. Arch., Bd. 28, p. 337 et seq.), whose essay opened the path to a correct understanding of the anatomy of the pock. Bärensprung, as is well known, attributed a partitioned structure only to confluent pocks, while denying it to the other varieties. The older authors considered this reticular structure to be fibrinous.

"*umbilicus*" is seen. This central depression usually occurs pocks in which a hair follicle or the duct of a sweat gland can be seen. Since the epidermis is continuous with the sheath of the hair follicles, when a vesicle is formed about such a follicle its centre will be held down, and the surrounding epidermis will be more elevated than in the portion continuous with the hair follicle. The ducts of the sweat glands have the same relation to the epidermis and act in the same way in holding down the centres of the vesicles (Rindfleisch). In a pock where there is neither hair follicle nor sweat gland it is evident that some more resistant portion of the tissue may in the same way hold down the centre of the wall of the vesicle. Auspitz and Basch explain the formation of the umbilicus in another way. They teach that the periphery of the pock swells more rapidly and thus becomes more voluminous than its centre.

When the pustule is fully ripe the umbilicus disappears by the stretching or destruction of the tissue which held down the centre of the vesicle. When desiccation commences, an umbilicus may again appear from the earlier drying of the centres of the pustules. This also takes place in pustules in which there was before no umbilicus.

When the pock is fully ripe,. the subjacent papillæ, which were at first swollen, become flattened. This condition may remain after the scabs have fallen off, and then give rise to shallow depressions in the skin. But these depressions are quite different from cicatrices. In confluent variola, sometimes in the discrete form, and occasionally in varioloid, the papillæ beneath the pustules become involved in the inflammation. Pus cells accumulate in these papillæ, compress the blood-vessels, and produce partial or total necrosis of the tissues. This condition is indicated by great swelling and congestion of the skin around the pustules. We find in the latter, besides pus cells, fragments of the destroyed papillæ. But some portions of the papillæ may still remain alive, and thus hold the scabs on for a long while. The extent of the ulcers formed by this destructive process determines the extent and shape of the cicatrices.

Of the different varieties of pocks enough has been said in treating of varioloid. I have convinced myself that in "variola verrucosa" the wart-like papules are produced by an early growth of the papillæ.

There is nothing specific in the formation of the hemorrhagic pocks. Erismann,[1] Wagner,[2] and Wyss[3] have proved that the bleeding has no connection with the hair follicles. The hemorrhagic eruption is seen in different individuals in different stages of development, according to the period of the disease at which the patient has died. The only difference between the hemorrhagic pocks and the ordinary forms, in corresponding stages of the eruption, is that their contents are bloody instead of serous or purulent. Besides the hemorrhagic pustules we usually find larger or smaller hemorrhages in the tissue of the cutis. In the less severe cases the papillæ are sometimes free from hemorrhages, and the latter are only found in the layer beneath; while in the severer forms all the layers of the cutis and even the subcutaneous fat may be infiltrated with blood. This last condition belongs especially to purpura variolosa, in which the patient dies before a pustule is formed. In this variety of variola the hemorrhages reach their greatest size, they may even be continuous over a large part of the surface of the body. Wagner has rendered it probable that the bleeding does not take place by rupture of the blood-vessels, but by transudation of the blood through their walls (diapedesis). No definite changes in the walls of the vessels to account for these changes have yet been discovered.

The *internal organs* undergo different changes in variola vera, in purpura variolosa, and in variola hemorrhagica, as Ponfick has very recently shown.

On the *mucous membranes* in variola vera, we find specific pustules, diffuse purulent infiltration of the middle epithelial layer, and catarrhal, croupous, or diphtheritic inflammation. The intensity and extent of the changes are often in direct relation to the changes in the skin, and not infrequently are developed before the latter.

Those mucous membranes are most frequently attacked which are the most exposed to contact with the air. In the nose the process is almost constant, and is hardly less so in the mouth and tongue. The tongue is frequently deprived of its epithelium over large tracts. The tonsils, the soft palate, the posterior nares and pharynx, and the lower ends of the Eustachian tubes are frequently swollen and infiltrated with pus.[1]

---

[1] Sitzungsbericht der Wien. Acc. 1868.

[2] Die epithelialen Blutungen. Arch. der Heilk. Bd. IX.

[3] Arch. f. Dermat. und Syph. Bd. III., S. 529.

In the trachea pustules are found as far down as its bifurcation, and at this point they are often numerous, sometimes confluent. The pustules may even extend down into the bronchi of the second and third order (Wagner). The mucous membrane is, at the same time, swollen, bluish-red, and often partly covered with a dirty-gray matter. The small bronchi exhibit various grades of catarrhal inflammation, and usually contain yellowish, bloody, or thin mucus. Catarrhal pneumonia is also of frequent occurrence, and may be succeeded by pulmonary phthisis.

In the œsophagus pustules are formed, but almost exclusively in its upper portion. In the stomach and intestines we only find catarrhal inflammation and small hemorrhages. In the intestines there may be ulceration of the follicles, with swelling of the mesenteric glands. It is very doubtful if real pustules are ever formed in the stomach and intestines, although they have been described by the older authors (Robert, Epidemic in Marseilles). They are only seen in the lowest part of the rectum, close to the anus. In the vulva and vagina there may be pustules or diphtheritic inflammation. In the urinary bladder pustules are never found, nor in the urethra, except close to the meatus.

True pocks on *serous membranes* are fables belonging to antiquity; but congestion, inflammation, and ecchymoses are common. The pleura is frequently affected in this way, meningitis occurs rarely, peritonitis probably not at all.

The *liver*, the *kidneys*, and the *spleen* undergo morbid changes of great consequence. Concerning these changes we find somewhat various statements by different authors. We find sometimes granular swelling of the liver and kidneys, sometimes acute fatty degeneration resembling that produced by phosphorus poisoning. In other cases these organs may be found unchanged, either when death takes place so early that there is not time for degenerative changes, or so late that the cells have passed through the condition of granular swelling and returned to their normal condition. Fatty degeneration is the more advanced condition in which granular swelling may terminate. In pustular variola the bile is usually pale and thin.

---

[1] L. c. This author gives us valuable data regarding the condition of the ear in variola, based on 168 cases. He found morbid changes in ninety-eight per cent. of this number. Pustules were present almost always on the external ear, less often at the beginning of the cartilaginous portion, while in the bony portion and tympanum only hyperæmia and swelling were found. These latter changes were also sometimes found in the middle ear, where pustules are never formed.

In the hemorrhagic variety of pustular variola I have found such degenerations of the liver, kidneys, and spleen as the rule; but in purpura variolosa Ponfick has shown that the absence of these lesions is a well-marked characteristic, a fact confirmed by my own observations. In this variety of variola the liver is usually of normal size, of dark color, and of very hard consistence.

In variola vera the spleen is much swollen in those who die early in the disease, and its pulp is soft and of a light red color. In those who die later it is more frequently unchanged. In the purpuric variety the spleen is small, hard, dirty, dark red, sometimes with large white or yellowish follicles (Ponfick).

It seems to me wrong, on account of these differences in the condition of the abdominal viscera in variola vera and purpura variolosa, and on account of the hemorrhages, to class these two varieties as distinct diseases. In purpura variolosa the patients die very early, so that we see lesions belonging to a very early stage of the disease. If we obtained autopsies in an equally early stage of the disease in variola vera we should see different conditions from those to which we are accustomed.

When the liver and kidneys are far advanced in fatty degeneration the *walls of the heart* are yellow, flabby, and brittle. But in purpura variolosa the organ is contracted, firm, and brownish red (Ponfick).

The *brain* and *spinal cord* are usually unchanged, although sometimes they are congested and œdematous.

In the hemorrhagic form of variola, besides the lesions mentioned, we find large or small hemorrhages in nearly all the v[illegible]cera. We find ecchymoses in the serous membranes, a[illegible] bloody fluid in their cavities. Very large hemorrhages may b[illegible] found in the loose connective tissue of the mediastinum, of th[illegible] pelvis, beneath the peritonæum and beneath the capsules of th[illegible] kidneys. Some authors are inclined to connect the hemo[illegible]rhages about the kidneys and beneath the peritonæum with th[illegible] pain in the back, which is so severe in some patients.

We may find hemorrhages in almost all the mucous mem[illegible]branes. They occur throughout the respiratory passages, fro[illegible] the nose down to the bronchi. They are also found in the pharynx, œsophagus, stomach, colon, and rectum, less frequently in the ileum (Ponfick).

In the mucous membrane of the uterus and Fallopian tubes, and in the parenchyma of the testicles hemorrhages are of frequent occurrence. The kidneys usually escape, but the pelves, calyces, and ureters are almost constantly the seat of large extravasations of blood.[1]

The liver, spleen, brain, and spinal cord are seldom affected in this way. In the lungs infarctions may be found, but are usually of small size.

Zülzer describes as of frequent occurrence hemorrhages in the sheaths of the nerves, and explains in this way some of the nervous symptoms. Wagner, however, did not find this lesion either frequent or extensive.

DIAGNOSIS.

The diagnosis of variola seems, *à priori*, a very easy one, and in well-marked cases with pustular eruption it is so. But in the less severe cases, especially in varioloid, when the eruption is scanty and undeveloped, the diagnosis becomes more difficult. The disease may then be confounded with syphilitic eruptions and with acne pustulosa, especially when there is no history of the previous condition of the patient.

Even more difficult, and sometimes impossible, is the diagnosis during the initial and eruptive stages of variola. And yet during these early stages the diagnosis is very important, for the patient is able to communicate the disease to others, and a failure in detecting it may be followed by disastrous consequences.

---

[1] Compare *Unruh*, Arch. der Heilk. Bd. 13, S. 289.

It is of great assistance to find out, in the first place, whether variola prevails in a given locality, and whether the particular individual has been inoculated, properly vaccinated, or has already had small-pox. We should also inquire whether the person has been exposed to contagion. Of the manifold ways in which such an exposure is possible, we have already spoken in detail in the section on ætiology.

If now we pass to the consideration of the first stage of variola we shall find that in many cases, especially of varioloid, the early symptoms are so vague, undetermined, and various that no diagnosis is possible until the characteristic eruption appears.

If we pass by these irregular forms, and consider the more intense and regular symptoms of the early stages, such as are usual in variola, and frequent in varioloid, the question arises, How is the diagnosis to be made?

In such cases I believe that by a careful observation and consideration of all the circumstances, we can often arrive at a definite opinion. There is only one pathognomonic symptom, and that is present only in the smaller number of patients. It consists in the already mentioned hemorrhagic initial exanthema, situated principally in the triangle of the thigh. The maculæ and diffuse erythemata without hemorrhages are not of themselves characteristic. Measles is the disease with which variola is most frequently confounded, and there is even an erroneous idea that there may be a combination of these two diseases. Let us then see what are the points of difference between them.

In *measles* we find, in the early stages, catarrh of the bronchi, conjunctiva, and nose, a condition which only comes on in a later stage of variola. The appearance of the skin during the stage of eruption may, taken by itself, give rise to doubt. We may lay down the rule, that in measles the maculæ are, from the beginning, larger than those of variola, and that they are developed almost simultaneously on the back and face; while in variola they begin on the head, and descend step by step downwards to the back. But this rule is only of much value in the regularly developed cases. Much more important is the degree of fever. In variola, during the initial stage, the temperature ordinarily rises to 104.9°-105.8°; while in measles during the corresponding period, it seldom exceeds 102.2°-104°. It is also characteristic of variola that soon after the eruption appears the temperature falls; while in measles it continues the same, or even rises. This peculiarity distinguishes variola from the other exanthematous fevers, and especially from *scarlet fever*. In this latter disease the early appearance of sore throat helps in the diagnosis. In purpura variolosa, however, the intense red color covering the back before the hemorrhages appear, may closely resemble scarlatina. And even after the hemorrhages appear it may be doubtful if the case is not an example of hemorrhagic scarlet fever. And the case may be still further obscured by the uncertain character of the temperature in purpura variolosa.

The diagnosis between *exanthematous typhus* and variola is, in their early stages, sometimes very difficult. The course of the fever during this period is hardly a guide at all. We have in both diseases the same rapid increase of temperature, and often the same maximum. But when the eruption appears the temperature falls in variola and does not in typhus.

In like manner the first attack of *relapsing fever* may resemble the initial stage of variola. The course of the fever may be the same in both diseases until the eruption appears. Sometimes, also, there may be doubt whether a case is one of intermittent fever or of the initial stage of variola. But in such a case the lapse of twenty-four hours is usually sufficient to remove the uncertainty.

In the case of *typhoid fever* mistakes are less frequent. The rise of temperature in typhoid fever is regular and characteristic. Enlargement of the spleen occurs in both diseases.

The increased frequency of respiration, the chill, and the fever in the initial stage of variola may perhaps mislead and make one think of *pneumonia*. But in such a case physical examination will remove all doubts, except in cases of circumscribed hepatization without physical signs.

The initial stage of variola is not likely to be mistaken for *acute miliary tuberculosis*. In the latter disease the ophthalmoscope may assist in the diagnosis.

Meningitis may resemble quite closely the initial stage of variola. In both diseases we have intense headache, vertigo, delirium, coma, and convulsions. Basilar meningitis, however, may be recognized by the local symptoms belonging to it. Cases of meningitis of the convexity extending over both hemispheres, and without localized symptoms, may give rise to much doubt. Cerebro-spinal meningitis may usually be distinguished by close examination, and by its ætiology.

From the different forms of *ephemeral fever* the initial stage of variola can usually be distinguished by the higher temperature. But in the irregular forms of varioloid the distinction is sometimes difficult. In the same way, the early stages of varioloid may be mistaken for *acute catarrhal gastritis;* while in a regular case of variola such an idea would hardly occur.

To mistake the severe lumbar pain in the early stage of variola for simple *lumbago*, is an error almost unpardonable.

In pregnant women we must take care not to confound the pains belonging to the initial stage of variola with *labor pains;* and, on the other hand, we must not forget that abortion and miscarriage are frequent results of variola.

PROGNOSIS.

The prognosis of variola must be considered in several different aspects. We must first regard the disease in general—its varieties and its different epidemics—then the separate cases and symptoms.

It is well known that small-pox was formally more dangerous and more dreaded than it is at present. In former centuries no disease was more dreaded, or destroyed more victims. It has been calculated that in the last century from 7–12 per cent. of all the deaths were due to variola. At the present day, in countries where vaccination is regularly practised, the proportion has fallen to 0.7–1 per cent.,[1] and would probably fall even lower if revaccination were more systematically and generally practised. The best proof of this is, that among infants, in places where vaccination is compulsory, the mortality from small-pox is almost nothing, while before Jenner's discovery, on an average, one-tenth part of all the children died from this disease. It is only after the age when the protection of the first vaccination is exhausted, that, at the present day, we see the disease becoming formidable. When, at this age, revaccination is compulsory, as in the Prussian army, its prophylactic effect is as great as that of the first vaccination.

---

[1] In Prussia, according to *Engel*, the mortality from small-pox in the years 1816–1860 was 0.7 per cent. of the entire mortality.

But though, at the present day, the mortality from small-pox is so much diminished, yet we find differences in the intensity and extent of different epidemics. That even in the worst epidemics the mortality is so much less than formerly, depends on two conditions, both intimately connected with vaccination. First, the immunity of vaccinated children from the disease; second, that among adults varioloid is much more frequent, and even variola runs a milder course. From varioloid alone healthy adults hardly ever die. The few deaths which do occur from it are in persons weakened by age or disease. Since, then, the mortality is almost exclusively dependent on the existence of variola vera, we may state that in general the mortality of a given epidemic is less in proportion as the number of varioloid cases is greater.

In variola vera the mortality is still very great; in some epidemics (for example the one in 1870–71) equal to that of former years. In some epidemics the mortality is increased by the larger number of hemorrhagic cases. The well-marked cases of this variety are usually fatal. Purpura variolosa is always fatal, while variola hemorrhagica pustulosa, in exceptional cases when the hemorrhages occur late and in small quantity, may terminate favorably.

Of 850 patients under my care 235 suffered from variola vera. Of these 46 were hemorrhagic, and all died. Altogether there were 99 deaths, about 42¼ per cent. If the hemorrhagic cases are excluded, the mortality of the other varieties was 28¼ per cent. This is a high percentage, and was due to the character of the patients—hospital cases from the poorer classes. An average percentage can only be given approximatively; it varies between 15 and 30 per cent.

At the present day some epidemics are severe, others are mild. The principal reason for the periodical outbreaks of the disease is the neglect of general vaccination and revaccination.

The mortality is usually less at the end of an epidemic than at its commencement. But the maximum of the extent of an epidemic, and the maximum of its mortality do not always correspond. More frequently the mortality is greatest at the time when the extent of the disease begins to decrease.

The season of the year has some influence on the course of epidemics; they are generally more dangerous in summer than n winter. Fortunately we no longer have many opportunities of confirming this experience of the older observers.

The prognosis of individual cases depends on a great variety of conditions. The age of the patients is of much importance. n infancy the mortality is enormous. Even including chilren up to ten years old, the mortality in the epidemics oberved by me was as high as fifty-eight per cent. In old age lso the prognosis is worse, even as early as the fortieth year. n adult life the prognosis is worse in women than in men, for in he former pregnancy, childbirth, abortions, and miscarriages re dangerous complications, and often develop the hemorrhagic orm of the disease. It is thought by many that the mortality s somewhat greater in women than in men, independently of hese complications. In men, however, intemperance adds to he fatal cases. Drunkards are very apt to suffer from hemorhagic variola, and delirium tremens is a very fatal complication. n other cases the viscera are so much altered by chronic alcoolism that the patients die from degrees of the disease which ther persons pass through safely. Overworked and badly ourished persons also seem to me more prone to die from this isease.

The constitution and physical condition of the individual also ave their effect on the prognosis. Robust and healthy persons will naturally endure a much greater degree of disease than hose enfeebled by scrofula, syphilis, or other chronic diseases. Persons who are convalescing from acute diseases, such as yphus, pneumonia, etc., die easily.

The question next arises how far we can regulate our progosis by the character of the disease and the separate symptoms.

The severity of the initial symptoms is not a safe guide for the prognosis. This stage has no direct relation to the intensity of the succeeding stages. Sometimes a very severe initial stage passes on into a light form of varioloid. If, however, the initial stage is mild, we may reasonably look for a moderate eruption. Very severe and continued lumbar pain is usually considered a bad symptom, and it does in fact frequently precede the hemorrhagic form of the disease. In my opinion the initial exanthema does furnish a prognostic sign of some importance. The assertion that it only precedes varioloid is not true. According to my experience it is rather the pure erythematous eruptions, after which we may expect varioloid or light variola. The number of the pustules is then frequently in inverse proportion to the intensity of the initial exanthema. On the other hand, if the initial exanthema has the hemorrhagic form, especially if it appears in the triangles of the thigh and arm (axilla), then we may almost always expect variola vera.

In the stage of full development the character of the pustular eruption determines the prognosis. Varioloid is very seldom fatal; confluent variola is very dangerous; semi-confluent variola, or discrete variola with very numerous pustules, are of doubtful result. Any one with large experience of small-pox must be convinced that many patients die simply from the dermatitis. In the confluent and other severe forms death often takes place during the stage of suppuration. In other cases the patients survive until the period of desiccation, and then die from inanition, or from furunculosis, erysipelas, or some other severe complication.

Next to the lesions of the skin those of the mucous membranes are of the greatest importance in prognosis. Diphtheritic affections, œdema of the glottis, bronchitis, and pneumonia are all dangerous complications. In infants the pustules of the mouth and pharynx may seriously interfere with swallowing.

Symptoms of grave cerebral disturbance, delirium, convulsions, and coma are dangerous. They may induce sudden death with apparent collapse.

The frequent complications of small-pox add to the mortality of the disease according to their character and intensity. We must never neglect to watch closely for them, as the patients frequently do not complain.

When variola terminates in recovery, the duration of the disease varies with its intensity and with accidental relations. In varioloid the duration varies from a few days to a month. Variola vera, even in the milder cases, hardly terminates in recovery before five to seven weeks. The duration of the confluent, semi-confluent, and severe discrete forms can hardly be reckoned beforehand. The numerous complications and sequelæ protract the disease even as long as several months. Many patients, without any severe local affections, are so prostrated that their recovery is very slow. This is said to be especially the case with the hemorrhagic patients, although in my own experience these latter cases all died.

TREATMENT AND PROPHYLAXIS.

The office of the physician in regard to small-pox is manifold. Besides the treatment of the disease in its various stages and varieties there is much to be done in the way of prophylaxis.

Against the spread of small-pox we possess a certain and eliable barrier in vaccination, by the use of which the disposiion to the disease can be almost entirely destroyed.

We will speak first, however, of the special treatment.

Are we able to exert any influence on the disease in the early tage preceding the eruption? Is it possible in infected persons during the stages of incubation and invasion to cut short the disase or to modify its course? Many attempts have been made o answer these questions affirmatively, but as yet without much esult. The first idea was vaccination, and this was employed by some in the ordinary way; by others subcutaneous injections of vaccine lymph have been made, it is said with good results.[1] must, however, advise great skepticism regarding these assertions. Of the subcutaneous injection of lymph I have no experince; but that ordinary vaccination during the stages of invaion and incubation cannot stay the disease, has been proved to me by chance observations and direct experiments. On the contrary, I have seen, in cases in which vaccination was practised after infection with variola, vaccine pustules and small-pox pustules developed side by side. It is, in my opinion, very doubtful whether vaccination can even render the course of the disease milder.

---

[1] *Furley*, Lancet, May 25, 1872.

The old attempts to cut short the disease by sweating, vomiting, purging, blood-letting, etc., are hardly mentioned at the present day. On the contrary, such remedies are used with caution, even for the relief of symptoms.

The assertion lately made (Stiemer), that by large doses of quinine given during the stage of invasion the course of the disease can be shortened and modified, is contrary to my large experience.

We can do nothing during the stage of invasion except to regulate the condition of the patient and treat special symptoms. The patient should be kept in a large, well-ventilated room, at a constant temperature of 60°-67° F. During extensive epidemics the large number of patients may render this impossible. There is still a popular idea that in small-pox and the other exanthemata there is some special virtue in keeping the patient hot and sweating, and we may sometimes find difficulty in overcoming this prejudice. The patient should be kept quiet in bed and given easily digestible food. Pure water is the best beverage, but lemonade, acid and mucilaginous drinks and Seltzer water, with or without milk, may be given. High grades of fever may render necessary the use of quinine, or digitalis, or of cooling baths or sponging. If the headache is severe and the face flushed, iced compresses and ice-bags usually afford relief. Pieces of ice and Seidlitz powder may be of use to relieve the vomiting and retching. When these are not effectual I have seen good results from the use of aq. amygdalarum and hypodermic injections of morphine.

When the eruption appears, the measures to be adopted will vary according to the form of its appearance.

The milder forms of varioloid usually demand no interference, and in some even the patient hardly needs to be kept in bed. In severe variola, on the contrary, the eruption itself, the symptoms, and the complications demand our attention.

It is unnecessary for us to speak of the specifics which from antiquity to the present day have been advertised and forgotten. The last of these specifics is the antiseptic, "parasite-killing" medication, for which carbolic acid is the fashionable agent.[1]

The methods of treatment proposed for the eruption are numberless. They are especially directed to the eruption on the face. Some endeavor to treat the separate pustules. An old plan, handed down from the Arabs, is to open the pustules and evacuate their contents, and this plan still has its advocates; but its only effect is to make the patient rather more comfortable. Some again cauterize each pustule after opening it; but this is hardly possible in confluent small-pox, and hardly necessary in the very discrete forms. The end aimed at by these procedures, to prevent the formation of deep cicatrices, is never attained with any certainty. If the pustules are superficial their scars will be slight; if the papillæ are involved, no amount of caustic can prevent the loss of substance.

Other attempts have been made to effect a coagulation of the contents of the pustules, or to abort them before they reach the vesicular stage. For this purpose the tincture of iodine has been much employed. I have no personal experience of its use, but reliable authors (Martius, Eimer, Knecht) say that the results of painting the face with it during the period of eruption are very striking. In an analogous way, during the same period, solutions of nitrate of silver may be employed with benefit. Zülzer believes that xylol given internally coagulates the contents of the pustules and cuts short their development. At the present time a definite opinion as to the value of this procedure cannot be given.

French authors have thought that mercury has a specific effect on the pustules, and have employed it as an ingredient in various salves. The principal value of this method is in the use of the fat, and simple inunctions of oil are equally beneficial. Mercury has also been used in the form of plasters, especially the emplast. de Vigo, with which the face was covered as with a mask. The good effects obtained by this plan seem to be due to the gentle pressure exercised on the face, and the action of the mask as an emollient and a cover. The same effects are produced by other indifferent plasters, and by lint soaked in oil or glycerine. Painting the face with collodion, as advised by Aran and Valleix, has not been successful in my hands.

---

[1] Thus we find an author claiming that by washing the skin with carbolic soap during the stage of eruption the disease was aborted, and supporting this claim by an experience of five cases!

According to my experience all these methods are far inferior to the use of cold compresses. I believe cold and moisture to be the most efficient agents in the treatment of the eruption. In all severe cases the application of iced compresses to the face and hands, or to any parts where the eruption is abundant, is to be warmly recommended. The severe pain is diminished, the swelling and redness of the skin are lessened, and the patients are much relieved. But no modification of the copiousness and development of the eruption is obtained by this plan either.

In the same way the intense pain often felt in the hands and feet may be relieved by wrapping them in cold cloths. The prolonged use of lukewarm hand- and foot-baths may also relieve this pain. I have not seen much benefit from the use of cataplasms. If the odor is very bad, carbolic acid, chlorine water, or other antiseptics may be added to the water.

In the stages of invasion and eruption, immersion baths may be beneficial. But their effect is not as marked as in typhoid fever, nor do they exert any influence on the course of the disease. In the period of suppuration it is very difficult to get the patient in and out of the bath, nor does the latter afford much relief. Cold compresses and sponging of the body are more easily used.

When the mouth and pharynx are much involved, astringent gargles are indicated; of these I recommend a weak solution of the liq. ferri sesquichlor. To relieve the pain and difficulty in swallowing, mucilaginous drinks, decoctions of althæa, etc., may be of service, as may also chlorate of potash, diluted chlorine water, and weak solutions of iodine. Sometimes antiseptic gargles, carbolic acid and permanganate of potash, may be of value. When there is acute œdema of the glottis we may give an emetic, if the patient is strong enough, or we may employ local scarifications or tracheotomy.

When the disease is fully developed the diet should correspond to the degree of the fever and the condition of the patient's stomach. If the patients are weak, bouillon with eggs, Liebig's beef-tea, and even wine should be given. The condition of the mouth may render many fluids objectionable, on account of the pain they give. In varioloid the appetite may be good in the period of eruption, and such patients should not be kept on an absolute fever diet.

Complications are to be treated according to general rules.

General blood-letting is usually badly borne in small-pox, and may produce sudden collapse or give rise to the hemorrhagic diathesis.

In severe delirium and in delirium tremens the chloral hydrate would be of great use if it were not for the affections of the pharynx and larynx; but these affections may be dangerously rritated by this drug. I have seen acute œdema of the glottis produced in this way. The chloral should be given therefore by the rectum (chloral, from one and a half to two drachms; gum arabic mucilage, water, each eight fluid ounces), or we may use bromide of potassium with opiates, or the latter alone, especially subcutaneously. Patients who are delirious need close watchng, or very disagreeable accidents may occur.

If the patients are weak, or fairly in collapse, quinine, camphor, wine, or alcohol should be given. A very good preparation is the Stokes' Cognac mixture: Best brandy, distilled water, each two fluid ounces; the yolk of one egg; syrup, one luid ounce. A tablespoonful every two to three hours.

During the stage of decrustation warm baths employed every day, or every other day, give great comfort to the patients, and assist in the falling off of the crusts. Inunctions with any kind of fat will alleviate the itching.

Abscesses in the subcutaneous tissue must be opened early and freely, for if the skin over them is too much stretched and hinned, it is a long while in returning to its normal condition.

When the crusts have fallen off we can do nothing to prevent he formation of cicatrices. But in variola verrucosa I have used with great benefit the tincture of iodine, painted on, as a remedy against the warty nodules which are left on the face.

We are almost powerless against the hemorrhagic form of small-pox. Acids, quinine in large doses, ergot, liq. ferri sesquichlor. are of no effect. Tonics and irritants also, although they have been warmly recommended, do but little good. When there are hemorrhages from the different cavities of the body, we must employ the different styptics, injections of ice-water, cold compresses, or tampons, although their beneficial effect is very slight. Transfusion, also, from which so much was hoped, has as yet disappointed us. But I should advise further attempts with this method.

Prophylaxis.

The prophylactic measures against the spread of small-pox are much more important than the treatment of the disease. The measures to be adopted are founded on an exact study of its ætiology, and of this we have already written at length.

In the first place, the sick should be strictly isolated, a plan not always easy to carry out. Isolation is proper even in the stage of invasion, for the disease is then infectious. Among the poorer classes, who live in crowded rooms and houses, we should, if possible, remove the sick to hospitals. The nurses and attendants should not be allowed to see other persons, or, if they do, should change their clothes and be thoroughly aired and cleaned. Physicians, also, should remember that they may carry the contagion.

Far more important, however, is it that every one likely to be exposed to the contagion should be at once revaccinated. This should be done even to persons who have been already exposed to contagion, as it is always possible that they may not have been infected, although exposed.

Patients who have recovered should not be allowed to see other persons until all the crusts have fallen off, for these latter are very apt to carry the contagion.

The bodies of those who have died from small-pox are capable of conveying the disease, so are the clothes, beds, and other effects of the sick, and the contagion may cling to them for a long time. All such articles are to be destroyed, or disinfected by heat, chlorine, sulphur, or long exposure in the open air.

Vaccination.

All attempts to check the spread of the disease by isolation and disinfection are of but little value in thickly populated countries and cities, for such attempts can never be completely carried out.

So deeply was this felt that inoculation of the disease itself was first resorted to as a prophylactic measure. Afterwards this unsatisfactory procedure was replaced by Jenner's discovery of vaccination. By vaccination the two great disadvantages of inoculation are avoided, the danger of the procedure,[1] and the fact that inoculated persons can communicate small-pox to others.

We must recognize Jenner as the discoverer of vaccination, one of the most important advances in medicine. Although before him the cow-pox and its relation to small-pox had been recognized in different places,[2] and vaccination had even been practised in Europe, in isolated cases, yet to him belongs the honor of establishing vaccination as a recognized preventive of small-pox. The industry, the straightforwardness, and the critical acuteness of Jenner's investigations may serve as models. Jenner began in 1776 to study scientifically the traditional belief of the country people of his neighborhood, that cow-pox was a safeguard against small-pox. In 1796 he made his first vaccination on man, and in 1798 he published his first important paper on the subject.

Jenner's experiments met, at first, with some opposition, but soon overcame it. In England, France, and Germany numerous experiments were made with vaccination, and it was found that small-pox could not be produced, even by inoculation, in vaccinated persons. In 1799 the first public institution for vaccination was established in London, in 1800 the new method was introduced into France and Germany, while now it is practised throughout the civilized world.

A word with regard to the *variola of the lower animals.* In various domestic animals, and in many others which come much in contact with mankind, certain eruptions are observed which have many features in common with the variola of the human species. Of especial importance among these are the *small-pox of horses*, and particularly the *cow-pox.*[1] The horse-pox is usually seen upon the foot-joints of horses, while the cow-pox, *variola vaccinia*, is almost exclusively observed upon the udder and teats of the cow. In both cases the eruptions, as a rule, are purely local, for exanthemata involving the entire body are of extremely rare occurrence in these animals, and are probably not of the same nature as the former. The horse-pox is very closely related to cow-pox, and Jenner states that the latter is sometimes due to the accidental transmission of the virus to the udder of the cow from the hands of a milker who has been taking care of horses affected with the variolous disease.[2]

---

[1] The mortality from inoculation was about two per cent.

[2] *A. v. Humboldt* asserts that among the mountaineers of Mexico the protective power of cow-pox against small-pox has been recognized and acted on for a long time. In Europe, *Sülzer*, in 1713, *Sutton* and *Fewster*, in 1765, called attention to this property of vaccine. A school teacher in Holstein, named *Plett*, vaccinated three boys in 1791. In Jenner's home, in Gloucestershire, it was a traditional belief among the country people that persons who acquired cow-pox by milking cows affected with the disease were safe from small-pox. It was on this experience that Jenner commenced his researches.

The horse-pox can very probably be inoculated upon the human subject with the same effect as vaccinia, and this practice is objected to merely because horses have other kinds of sorès upon the foot-joint, which might occasion disagreeable mistakes sometimes.

Respecting the origin of variola vaccinia, which is by no means a frequent disease, we have as yet no very certain knowledge. A "spontaneous" development is accepted by many, upon no very strong evidence, however. The inoculation of human small-pox lymph upon cows produces variola vaccinia, the same as after inoculation from the horse-pox, and if a susceptible person is vaccinated from a variola vaccinia acquired in this way, merely local pustules will be produced without any general eruption following. However strongly these circumstances speak in favor of the identity of human and cow-, or animal-pox, for which many writers very zealously contend, they do not absolutely prove it, and hence, for the present, we can only admit the probability of the supposition that variola vaccinia is small-pox, so modified by the nature of the soil that it retains its tendency to strict localization, even when re-engrafted upon the human species.

---

[1] Besides these two forms the sheep-pox is also of some importance. This is said to run a more severe course than the cow- or horse-pox. Moreover, pocks have been observed in goats, pigs, asses, dogs, and indeed in monkeys. The pocks that occur in any one of these animals appear to be capable of successful inoculation upon all of the others.

[2] From cows that had been infected in this manner Jenner made a number of vaccinations upon people, wherefore his enemies declared that he had performed his inoculations from bad cases of horse-pox.

Although, in accordance with the method of Jenner, vaccination was originally performed with cow lymph, vaccinia is now almost universally transmitted from one person to another through many generations, only the first individual in the series receiving the virus direct from the cow. The vexed question as to the superiority of this "humanized" lymph over he actual contents of the cow-pock has not as yet been settled. To attempt to decide the question at present would be njust to both sides. Two objections are urged against the use f cow lymph; first, it is claimed that vaccinations with it fail ore frequently than with humanized lymph; and secondly, that he local symptoms are much more violent than where the latter s used. The objection has been made to humanized lymph, on he other hand, that its protective power becomes gradually eakened after it has passed through several generations.[1] We ust reject all such assertions as are based upon general impresions, and are not supported by extended statistical investigaions. The investigations thus far made lack both exactness nd scope, for, unfortunately, in no country at present are vacination and revaccination so carefully managed, or under such erfect control as would be desirable. As the question now tands, there is no sufficient reason why we should give up the se of humanized lymph for true vaccine matter direct from the ow, and the former is much more easily obtained. We shall ecur later to certain objections to vaccination, which relate specially to the use of humanized lymph.

If we watch the processes which take place locally after vacination, we shall perceive in their course and development an nmistakable resemblance to the pock of true human variola. 'o begin with, we have here also a period of incubation, although of far shorter duration. Two, and still more often three, days elapse from the time of inoculation before the spot of efflorescence begins to develop. The first apparent symptom is a redness and slight swelling of the skin, which preserves the form of the original wound. By the next day the summit of the papule, which has meantime considerably increased in size, shows a little vesicle, filled with a clear liquid, which now gradually grows larger and larger. As the circumference of the efflorescence increases, an umbilication appears, like that in the genuine variola pustule, and this also assumes the shape of the original wound (round or elongated, according as punctures or incisions were made), and this is due to the fact that at the point where the wound was situated a firmer adhesion to the subjacent parts has taken place than in the peripheral portions, which latter are therefore able to extend considerably, as the amount of the contained fluid increases. The pustule attains its greatest extent about the seventh or eighth day, and at this period contains a clear liquid which, just as in the variolous pock, does not wholly escape as soon as a puncture is made, but oozes out in the form of a small drop. This is due to the fanlike construction of the meshwork, which is the same as in the true variola pustule, and is dependent upon the same causes. After the eighth or ninth day the contents begin to grow cloudy, and by the eleventh or twelfth the previously pearl-colored, somewhat pellucid pock has become entirely yellow, while in the centre, just at the location of the original wound, the crust formation has begun. While the pustule is at its highest development the surrounding skin is more or less swollen and red. The inflammatory areolæ of the separate pustules often run together, so that the latter then appear to rest upon an uniformly reddened and swollen base.

---

[1] Some have lately made the assertion that since Jenner's time the lymph has radually deteriorated, and in view of this supposed fact, Coste has quite recently ade the somewhat singular proposition, that vaccine lymph be taken from cases where ariola and vaccinia are simultaneously present in the same individual.

At this time fever occurs, which is proportionate to the intensity of the local symptoms, and is accompanied by the ordinary derangements of the general system which are usually associated with it. At the commencement of desiccation the local, inflammatory, and the general symptoms rapidly disappear. The drying begins, as already stated, in the middle of the pustule, commencing with a thin scab, which gradually increases in thickness and peripheral extent until about the nineteenth to the twenty-first day, and then falls off, leaving behind a scar which at first is slightly red, but afterwards is white and radiated, or striated. These scars, as should be distinctly understood, are in themselves not at all characteristic, and it is only from their situation and arrangement, and on account of their uniform size, that we are enabled to surmise an artificial origin.

In spite of the efforts of its opponents, no unprejudiced person at the present day can any longer be in doubt as to the efficacy and eminent practical value of vaccination. In countries where it has been introduced, and in a measure systematically carried out, the number, the intensity, and the extent of small-pox epidemics have been notably diminished, and in a manner which of itself renders the idea of mere coïncidence inadmissible. In this connection nothing could be more convincing than the exceedingly interesting and graphic account which Kussmaul[1] gives of the mortality from variola, in Sweden, during a period of one hundred years, in the latter half of which vaccination was universally practised. Moreover, for Germany, France, and England a somewhat similar decrease in the small-pox mortality might be demonstrated. If, notwithstanding all these proofs, we for the moment entertain the supposition, improbable as it is, that this decrease in the epidemics is a matter of mere accident, it at once falls to the ground as soon as we proceed further into detail. We see, first of all, that where vaccination is regularly practised in very early life, the mortality of children from small-pox, instead of being as enormous as amongst those not vaccinated, is almost nil. We notice further, that where the vaccination of adults, as for example in the Prussian army, is performed with regularity, epidemics of the disease no longer occur. With these facts before us the idea of mere coincidence is out of the question. The trial of vaccination in the Prussian army has conclusively demonstrated the efficacy of the measure, to test which we have only to compare the relative immunity of soldiers during great epidemics of small-pox with the mortality in classes of the same general age in the civil community where vàccination is imperfectly carried out.

---

[1] *Kussmaul*, Zwanzig Briefe über Menschenpocken- und Kuhpockenimpfung. Freiburg. 1870. We would very warmly recommend a perusal of this eminent work. It presents, in an unpretentious popular form, a mass of scientific matter combined with critical observations.

Although most persons after having had variola vera enjoy for the remainder of their lives a complete immunity from the disease, the protection which vaccinia affords against small-pox is only of a limited duration. The exact length of this period of insusceptibility cannot be stated ; in the first place because it always varies in different individuals, but especially because its own limits are never sharply defined—the disposition always returning, but very gradually. In general, the duration of immunity may be stated at from eight to ten or twelve years. In order to maintain a state of perfect immunity after this period a revaccination is required, and this should then be repeated at every expiration of the above-mentioned period throughout the remainder of life. It is only by the careful and universal performance of revaccination that we can insure the most complete effect of vaccination in permanently decreasing epidemics of small-pox. Were revaccination performed with even approximate thoroughness, the statistics of the mortality amongst those vaccinated, in comparison with those not vaccinated, which now occasionally supply an argument to the enemies of vaccination, would be far more favorable. At present, when the term "vaccinated" is used in any table of statistics, unfortunately we can only tacitly surmise that in the list of those properly so classed very many were included the date of whose last vaccination was very far removed, and consequently that their susceptibility had long since been renewed. Under existing circumstances it would be much more accurate to place opposite the number of those not vaccinated the number of those who had been successfully vaccinated within a comparatively recent period (say within eight years). I ascertained to my entire satisfaction, with regard to over one thousand small-pox patients whom I examined with reference to this point, that, although many of them had been vaccinated, it had been done either improperly or too long before the date of the attack ; not a single one met the strict requirements of an effective vaccination.

Besides inefficacy, all manner of directly injurious effects have been attributed to vaccination. That such effects may follow vaccination there can be no doubt, but it is idle to regard these as any sufficient reason for rejecting vaccination. We should rather endeavor to ascertain how numerous and how great the possible grounds of objection are, and to what extent they may be avoided.

But even vaccination, it is said, makes the children ill, and in many cases has, in fact, been the direct cause of death. If caution is exercised in vaccinating, that is, if the operation is not performed upon too young, feeble, or sickly children, nor during the period of dentition, nor at very unfavorable seasons of the year, the bad results will be so extremely rare that, in comparison with the advantages of the method, they will appear of trivial importance. Healthy children, if not too young, simply manifest, during the few days that the pustules are at their highest development, certain febrile disturbances of the general system, during which the temperature sometimes reaches 104° F. But these symptoms quickly pass away, as desiccation commences, without leaving any permanent ill effects behind. The occurrence of erysipelas, however, is something more serious. This may spread from the point of vaccination over a region of variable extent, and in certain cases may prove of very great danger. But, compared with the millions of vaccinations that terminate favorably, the number of cases which become dangerous or fatal in this manner is extremely small. Were we to neglect vaccination on this account, it would be about as wise as though we should refuse to travel by railroad for fear of possibly running off the track. Pseudo-erysipelas, lymphangitis, and inflammatory swellings of the axillary glands are of still more rare occurrence than vaccinal erysipelas.

Aside from the vaccination diseases proper, those objections to vaccination, based upon the supposition of its leading to permanent derangements of nutrition or of the general health, or upon the idea that it may afford an opportunity for the transmission of still other diseases than those mentioned above, have met with but slight acceptance.

In healthy children those disorders of nutrition or of the constitution which result from vaccination never remain for any length of time after the accompanying febrile symptoms have passed away. If just after this, however, the children should seem to be considerably affected, though even this is rare, they recover rapidly, and are generally quite well after a few days. But if, on the other hand, children who are already delicate, or have an hereditary disposition to scrofula, are vaccinated at an improper time, their doubtful condition of health at this tender age may thereby, as from any other febrile disease of like intensity, receive a permanent shock, and the scrofulous tendency may be thus hastened in its development. There is no doubt that in the latter case phthisical and tuberculous processes, which are so closely akin to scrofula, may now and then be developed. But in all this we discover no reason why we should abandon the practice of vaccination, but simply a motive for previously examining the condition of health of those to be vaccinated with the greatest exactness and circumspection. If, in case the children are delicate, or evince any special morbid tendencies, we wait until they are somewhat older and more strongly developed, vaccination may usually be performed upon them from the second to the fourth year without any injurious result. During small-pox epidemics delay is of course unjustifiable even in these instances. At such times the apprehension of the above accidents, which never happen indeed, except in a very small number of cases, is quite overshadowed by the threatening dangers of the disease.

Scrofula and phthisis are cited as amongst the very chief of those diseases which may be directly transmitted to healthy individuals by means of the lymph. But not the slightest evidence is adduced in support of this assertion. We have already admitted that in a small number of cases these conditions may arise indirectly, and where, in some other instances, these extremely common diseases have chanced to occur, the opponents of vaccination, partly blinded by their over-zeal, and sometimes not without design, have had recourse to the deceptive "post hoc, ergo propter hoc."

The possibility of the actual transmission of a disease through vaccination has thus far been demonstrated in but a single instance, and that is *syphilis.* Could the opponents of vaccination show that this occurs with any degree of frequency, or is with difficulty prevented, vaccination would thereby receive a severe blow. But here, fortunately, lies the weak point in our opponents' deductions. In the first place, those cases where the actual inoculation of syphilis has been verified are so exceedingly rare that the objections based upon them are consequently materially weakened. The force of these objections is still more impaired by means of the evidence, almost always present, that the unfortunate result was due to actual carelessness, or to an oversight easy to be avoided. *Nearly all of the unhappy occurrences of this sort are not the fault of vaccination, but of its improper performance.* Our experience regarding the inoculation of syphilis admonishes us, therefore, not to throw aside from mere timidity one of our most valuable safeguards from small-pox, but to endeavor to ascertain how we may avoid this undeniably serious accident. The most radical means to avoid it would be to vaccinate with lymph directly from the cow, and to this we should resort under certain circumstances. We have already spoken, however, for practical reasons, in favor of the ordinary use of humanized lymph, and in the great majority of cases can find in the danger of inoculating syphilis no sufficient reason for giving up this method. Of course great caution and care is very necessary in this matter. We are forced to confess that at present carelessness and negligence, especially in the procuring of vaccine matter, are too common. Particularly objectionable is the common custom of using for vaccination lymph which is brought into market, preserved in various ways, when nothing is certainly known as to its origin. Only such lymph should be used as either comes from a well-conducted institution, or is obtained by the physician himself directly from suitable cases. In the latter case the vaccine matter should not be taken from an adult if it can be avoided. Only in the most pressing cases is this allowable. Respecting the children, it is advisable to select lymph from such as are not too young, in no case before the expiration of five months, and still better after the first year. If congenital syphilis were present, it is to be supposed that some distinct manifestation of this would show itself within the first year, or, indeed, as a rule, within the first five months. It is furthermore advisable to vaccinate, when possible, only from those children whose parents' state of health is known, and on this account to avoid illegitimate children. The particular caution to examine carefully the entire body of the children from whom lymph is taken for the purpose of vaccination does not appear so superfluous as one might *à priori* imagine, when it is remembered how often "the busy practitioner" merely exposes the arm, or the upper portion of the body before taking the vaccine matter.[1]

According to an hypothesis of Viennois', it has been thought that the transmission of the disease might certainly be avoided, even in vaccinating from syphilitic children, if the clear lymph alone were used, with no admixture of blood. The lymph, according to V., is never the vehicle of the syphilitic poison, while the latter is invariably contained in the blood. That so absolute an assertion is untenable has been proved by experiments, and is confirmed by experience. It appears to be a fact, however, that bloody or cloudy purulent lymph is more dangerous than that which is perfectly clear. Moreover, we would call attention especially to the fact that not in all, indeed in but a small number of the cases vaccinated from syphilitic children, is there disease transmitted. This may partly be explained by reference to the above-mentioned qualities of the lymph (whether clear or mingled with blood), but more frequently the cause of the immunity is not apparent. It is not easy to solve this doubt, since experimental investigations are precluded, from the very nature of the case, and the few chance cases which are available to observation are fortunately extremely rare.

It is also frequently asserted that other, non-syphilitic eruptions arise in consequence of, or are transmitted with, vaccination. It is not uncommon to observe a macular or diffuse erythema, or even urticaria, making their appearance, usually just as the vaccine pustules are at the height of their development, or during their desiccation. The dependence of these upon vaccination cannot be denied, yet no reasonable person would oppose the latter on account of such insignificant occurrences.

---

[1] The case of Dr. H., of Holfeld, so often quoted by way of condemnation, offers an apt illustration of this point. The trial at that time revealed the fact that H. had simply exposed the arm of the syphilitic child from whom the vaccine material was taken.

The belief that healthy persons, who are not already predisposed to them, may be attacked by chronic cutaneous eruptions, especially eczema, in consequence of vaccination, is utterly unfounded. In those who are so predisposed there is no doubt but these eruptions may sometimes, though rarely, occur. The eczema then first shows itself generally in the neighborhood of the point of vaccination, and extends from there over a greater or less portion of the rest of the body.

An objection which shows a great want of discernment, but which has been frequently urged, is that since the general introduction of vaccination, as it is claimed, certain other epidemic diseases, particularly scarlet fever and measles, have become more frequent and more malignant. The absolute number of deaths from these diseases may have been somewhat increased, but this is by no means true of their relative number. This is readily explained by the fact that in consequence of vaccination a large number of individuals, who otherwise would have died of variola, remain alive, thus leaving a larger number for measles and scarlet fever to attack. A similar objection which has been raised in connection with typhoid fever, may be disposed of in the same way.

In regard to the performance of vaccination, a number of points are to be observed with especial care. Above all we should be very particular in procuring the lymph which is to be used, taking it only from perfectly healthy children, and from those vaccinated for the first time. We find, it is true, that the possibility of transmitting diseases in vaccination is far less than is generally supposed, and that we can assert this possibility with perfect certainty only of syphilis. But in a procedure which so many contend against, and which, especially in the eyes of the laity, is so objectionable, even the appearance of error must be most scrupulously avoided, not only for the personal interest of the physician, but in the interest more especially of a good cause. The early years of infantile life are threatened by a multitude of dangers, aside from this, and should any unfortunate accident occur, the laity are generally very eager to trace its source to the vaccination, and especially to circumstances connected with its performance. We have already mentioned that lymph taken from syphilitic children, and employed in vaccination, does not always transmit the disease, and Bousquet, who intentionally vaccinated with such lymph, has never observed infection follow. Concerning the proper age at which vaccination should be performed, and the necessity for a careful examination of the child from whom the lymph is taken, we have already said all that is requisite. Vaccination from adults should be avoided, and especially from those who have been vaccinated more than once, since in the latter case the lymph is much more apt to operate with uncertainty. But during great epidemics, when there is a lack of vaccine virus, necessity may oblige us to resort to even this doubtful source. This was the case in many places in the epidemic of 1870–71.

The best time for taking lymph from the child is on the seventh or eighth day after vaccination. We should select such pocks as are well developed, and it should be borne in mind that in order to obtain an abundant supply it is not sufficient simply to puncture the vesicle, but the epidermis over it must be incised for a considerable extent (on account of the fan-shaped structure). Before and after the seventh and eighth day it is much more difficult, if not impossible, to procure the lymph, and it is decidedly less reliable in its action.

It is always best in the performance of vaccination to carry the lymph directly from one arm to another. Often, however, this is impossible, and on account of the frequent necessity of waiting a long time after obtaining the lymph, before using it, its power of retaining its properties becomes of great service. With proper means of preservation the lymph retains its activity for a long time. The most unsuitable method for preserving it is that which was formerly very generally employed, namely, of allowing the lymph to dry upon little rods. A much better method, and one that is at present much more common, consists in preserving the lymph in capillary tubes which are hermetically sealed at both ends (by fusion or with sealing-wax). Müller has the great credit of having discovered the fact that by mixing vaccine matter with glycerine in certain proportions, the activity of the former is not diminished, so that we have here a means of increasing the volume of the lymph when the quantity is small or when there is an unusual demand for it. This discovery is of great value, since the certainty of success after vaccination, apart from the quality of the lymph, is dependent to a certain extent upon its quantity. The lymph and glycerine mixture appears to keep quite as well as the unmixed lymph. The objection frequently made, that vaccinia develops with less certainty and completeness after this method, I am able to refute from personal observation. I have never seen better developed vaccine pustules than at the Berlin Vaccine Institution, where such lymph is employed; and after my own numerous xperiments I can assert that the certainty of result is not at all ss than after the use of the pure lymph.

Müller usually mixes the lymph carefully with two parts of glycerine and two urts of distilled water (by means of a small brush in a watch-glass), and preserves e liquid in air-tight capillary tubes. This mixture, according to M., can be still rther diluted (one part to eight) without suffering any perceptible loss of its ficacy. Beyond this its activity is lessened in proportion to the degree of its dilu-on. Reiter[1] obtained a feeble action with dilutions consisting of one part of lymph two hundred of distilled water, and he ascertained that, as a rule, in order to otain any effect a greater quantity must be employed the more the virus is diluted. The direction generally given to avoid any admixture of blood with the lymph iginated in the idea, formerly entertained, that dyscrasiæ were capable of being ansmitted with the blood; but at the present time it is only with regard to syphi-s that any importance is to be attached to the precaution. Where this disease is it of the question the admixture of a drop of blood does not signify. Reiter's genious experiments have shown even that vaccination may be successfully per-ormed from the bearer of vaccine pustules using nothing but the blood, though the tivity of the latter is far less of course than that of the lymph, being about as one twelve.

As regards those who are to be vaccinated, no less caution is ecessary in their choice than in the selection of the children rom whom the lymph is taken. When small-pox epidemics do ot render vaccination imperative, it should be performed only n such children as are robust and not too young. They should e from four or five up to twelve months of age, according to circumstances, should not present at the time any other morbid symptoms, and not be teething. The period of weaning also is a particularly unsuitable time. A rigorous season of the year and times when epidemic diseases prevail among children should likewise be avoided if possible. In the case of weakly children and such as have a special morbid disposition to scrofula, phthisis, etc., it is generally well to postpone the vaccination until they have become stronger, since in such children vaccine, like any other intercurrent disease, is liable at the critical period of incipient development to occasion a derangement of nutrition which may be of long continuance, if not permanent.

---

[1] Bayr. ärztl. Intell.-Bl. 1872, No. 15.

In the vaccination of adults such great caution is of course unnecessary.

We now come to speak of the mode in which vaccination should be performed. It is generally done upon the arm, and usually on both sides. In girls just that portion of the upper arm (which, by the way, is of rather limited extent) should be carefully selected which will be concealed by the future evening toilet. The method, still much in vogue, of vaccinating children in the situation of nævi upon the face or some other conspicuous part, with the purpose of simultaneously accomplishing two ends, appears to me of rather doubtful utility.[1] In order to introduce the vaccine matter into the skin, a number of punctures or scarifications are made with a vaccinating needle or lancet, which has been previously moistened with the lymph. Where vaccine matter is to be had in sufficient quantity, it is purely a matter of custom whether punctures or incisions are made. If, however, the supply of virus is deficient, and it has been necessary to dilute it with glycerine to the utmost possible extent in order to make it suffice for the number to be vaccinated, then the method by incision is decidedly preferable. Reiter has established this point by convincing experiments. When lymph diluted in a certain way was brought into contact with the skin by means of punctures, incisions, or by rubbing it over a vesicated spot, the best effect was produced in the last case; from the punctures not a single pustule was produced, while after the incisions a few characteristic signs manifested themselves.

---

[1] In one case, to be more fully described elsewhere, a cicatrix of the face (resulting from vaccination of a nævus at the age of two years) was shown with great probability to be the starting-point of a lupus that developed at the time of puberty just in this place.

If vaccination is made with incisions, from four to six usually suffice for each arm. These should not be too close together, in order to prevent the pocks, and if possible their inflammatory areolæ also, from afterwards coalescing. Too deep incisions are inadvisable on account of the copious bleeding, by means of which the vaccine virus might be washed out of the wound. This danger, however, appears not to be as great as is generally imagined. The precaution not to cover the wound after vaccination until the blood has entirely dried, seems somewhat superfluous. The blood has often been intentionally wiped away from the wounds without the result of vaccination being at all impaired.

After the vaccine pustules have developed they should be relieved from any pressure or friction. Not so much because their destruction at a certain stage might lessen their protective influence, as for the sake of not adding any traumatic effect to the already existing inflammatory symptoms.

If no pocks are formed after vaccination, the attempt should soon after be repeated—in the course of the following month at least—with all the necessary precautions. The failure of vaccination may depend upon several reasons. In the first place, and this is especially true of revaccination, the individual in question at the time of the vaccination may not have been susceptible to the virus, and hence we may conclude that he was also not disposed to variola. Or else the vaccination itself was defective, either on account of some fault in its performance or from the use of inactive lymph. The latter possibility should be particularly borne in mind in case lymph was used that had been preserved for some time, instead of the vaccination being made directly from arm to arm.

In those cases, too, where at the point of vaccination, instead of the normal vaccine pustules, other lesions—macular erythema, pustules, vesicles, or abscesses (erroneously termed spurious vaccinia)—make their appearance, the repetition of the vaccination with other lymph, and if possible from arm to arm, is absolutely necessary. It will generally be found that the cause of failure is to be attributed to the quality of the matter employed, though in revaccinations the reason is quite as apt to be due to a want of susceptibility to the vaccine virus.

•

A weapon of defence against the scourge of small-pox so powerful as vaccination should not be left to the pleasure of the individual, but the State has the right and the duty to look after its most thorough performance. As in other spheres, where for the attainment of a result perfect co-operation is requisite, so our experience in regard to vaccination teaches that instruction and admonition alone are almost powerless in the struggle against negligence, deficient sense of duty, and evil disposition which oppose this useful measure at every step. Those acquainted with the subject therefore have long since been of the opinion that to the attainment of the desired end *legal compulsion* is absolutely necessary. The authority of the State to effect this is unquestionable in the minds of those free from prejudice, for the effectiveness of the method is beyond all doubt. Indeed, even since vaccination has been very imperfectly employed against the decimating plague, the average length of life has undergone an evident increase. Compulsion is furthermore justifiable on the ground that a certainty of success is impossible so long as a few individuals are not subjected to the operation, since from the extraordinary contagiousness of small-pox these become conveyors and diffusers of the poison. Even if vaccination were to be considered in the light of a sacrifice, the State might *under certain circumstances* demand this from the few in the interest of the community, and upon this basis establish a large number of State institutions. But even the selfish pretext that vaccination is injurious becomes, as we have seen, in so far untenable as the great number of the formerly accepted dangers of vaccination have been shown to be wholly imaginary, and *the unquestionable harm which only rarely occurs can be certainly avoided by means of caution and experience.*

We, therefore, greet with pleasure, in spite of its many imperfections, the law which the Imperial Diet has recently passed (April 8th, 1874) concerning vaccination. According to this law every child that has not already had small-pox must be vaccinated before the termination of the calendar year following the year of its birth, and every pupil in a public or private institution is to undergo revaccination within the year in which his twelfth birthday occurs. For the most convenient and cheapest methods of carrying out this plan, for the procuring of good lymph and its free distribution to physicians, as well as for accurately recording the results of vaccination, both in isolated cases and for general statistics, the law makes the most ample provisions. The paragraph of the previous statute which was designed to regulate the revaccination of adults at the outbreak of small-pox epidemics was unfortunately rejected by the Diet. To repair this mistake, and as soon as possible obtain the necessary addition to the vaccination law, is an important task for those conversant with the matter.

ERYSIPELAS.

(ROTHLAUF, ROSE, ST. ANTHONY'S FIRE.)

ZUELZER.

ERYSIPELAS.

560-1700. *A. Ellinger*. Dissert. de erysip. Leipz., 1560.—*Heurnius*, Diss. Leyd., 1596.—*Schoen*, Diss. Basel, 1605.—*Burmeister*, Diss. Basel, 1615. —*Schilling*, Diss. Lips., 1621.—*Küffer*, Diss. Argent., 1640.—*Mooeling*, Diss. ἐρυσιπελαγυμνασία. Tubing., 1621.—*Michaelis*, De rosas vero ac legitimo erysipelate. Leipz., 1655.—*Metzger*, Diss. historiæ erysipelatis. Tüb., 1666.—*Vehr*, Diss. Altd., 1667.—*Schenk*, Ordo et methodus tract. et cur. febr. erysipelat. Jen., 1666.—*Schneider*, Diss. Wittenb., 1568.—*Wedel*, Diss. Jen., 1682.—*Sydenham*, Oper. univ. Lond., 1685. p. 310.—*Dessali*, Diss. Leyd., 1694.—*Mappus*, Diss. Argent., 1700.—1701—1800. *Jacobi*, Diss. inaug. Erfurt., 1711.—*Zabel*, Diss. Lyon., 1717.—*Fr. Hoffmann*, De febr. erysipelacea. Halæ, 1720.—*de Pré*, Diss. d. Erys. (vulgo Rothlaufen.) Erfurt, 1720.—*Goelicke*, Diss. Frankf., 1736.—*Charleville*, De erysipelate pustuloso. Halle, 1740.—*G. G. Richter*, De erys. Diss. Götting. (Opuscula medica, 1744. I.)—*Juch*, Diss. de erysipelaceis inflammationibus. Erfurt, 1752.—*Aurivillius*, Diss. Upsala, 1762.—*Hermann*, Diss. de rosa. Argent, 1762.—*Monro*, The Diseases in the Brit. Military Hosp. in Germany. Lond., 1764.—*Schweder*, De febrib. erysipel. Gött., 1771.—*Dale*, De erysipelate. Edinburgi, 1775.—*Burean*, On the Erysipelas which is called St. Anthony's Fire. Lond., 1777.—*Gregory*, Lect. on Fev. and Inflamm. Edinb., 1777.—*Luther*, Diss. Erfurt, 1780. *Hellbach*, Diss. de erysip. Erfurt, 1780.—*Gulbrand*, Observationes de erysipelate. Acta reg. societat. med. Havn. I.—*J. J. Aerts*, Diss. Lovan., 1782. —*van der Belen*, Diss. Lovan., 1782.—*Gourlay*, Diss. Edinb., 1782.—*Kyper*, Inst. med. de erysip. Montpellier, 1783.—*Hoffinger*. De volatico s. de erys. erratico. Vindob., 1789.—*Cullen*, Méd. Prat. Trad. de Bosquillon. Paris, 1789.—*M. Culley*, Diss. Edinb., 1790.—*Ammon*, Diss. de erys. ejusque ab inflammatione diversitate. Hard., 1790.—*Thierens*, Diss. Lugd. Bat., 1790. —*Fowle*, De febr. erysip. Edinb., 1791.—*Gergens*, De erysipelatis febrisque erysipelatosæ causa materiali. Mogunt., 1792.—*Winkel*, De cognoscendo et curando erysipelate. Erl., 1794.—*Ferne*, De diversa erysipelatis natura. Frankf., 1794.—*J. P. Harmand de Montgarny*, Précis med. et curatif. de malad. éruptives, connues sous le nom de rose épidémique, qui regnent dans le département de la Meuse. Verdun, 1793.—*Engelhart*, Diss. Lundæ, 1797.—1800-1820.

Zimmermann, Prag. Vierteljschr. IV.—*Norris*, Med. Times and Gaz., Dec. 1852.—*Blake*, American Journ., 1852, July.—*Hamilton Bell*, Edinb. Med. and Surgic. Transact., 1853. (Tinct. ferr. muriat.)—*Balfour*, Monthly Journ., 1853. (The same.)—*Venot*, Journ. de méd. de Bordeaux 1853.—*Bennet*, New York Journ. of Med., July, 1853.—*Zuccarini*, Wien. med. Wochenschr. 1853. Nr. 4–7.—*Corson*, Transact. of the Med. Soc. of Pennsylvan. IV. 1854.—*Hasse*, Deutsche Klinik. 1855. Nr. 29.—*Avery*, Transact. of the New York Med. Soc., Americ. Journ. of Med., Oct., 1855.—*Velpeau*, Bull. gén. de thérap. 1855. (Sulphate of iron.)—*Lorain* (*Paul*), La Fièvre puerp. Paris, 1855.—*Todd*, Med. Tim. and Gaz., 1855. July 27.—*Bouchut*, Traité pratique des maladies des nouveau-nés et des enfants à la mamelle. Par., 1855.—*Gubler*, Mém. de la Soc. de Biolog. 1856. p. 40.—*Nathusius*, De erysipelate typhoide. Diss. Berol. 1856.—*Imbert-Gourbeyre*, Gaz. méd. 1857.—*Aubrée*, Thèse de Paris 1857.—*Barbieri*, Gaz. medic. ital. Lombard. Nr. 54. 1857.—*Arnott*, Medic. and Physic. Journ., March, 1857.—*Betz*, Memorabil. d. Heilk. II. 1857. 10. 13.—*P. Hinckes Bird*, Midland Quarterly Journ., May, 1857. Schmidt's Jahrb. Bd. 96. p. 179.—The same. On the Nature, Causes and Treatment of Erysipelas. Lond., 1858.—*Labbé*, De l'érysipèle. Thèse de Paris. Nr. 168.—*Lebert*, Virch. Arch. Bd. XIII. p. 371.—*Déchambre*, Gaz. hebd. 1858.—*Duncan*, Edinb. Med. and Surgic. Transact. V. I.—*L. Forrer*, (Winterthur), Ueb. d. versch. Formen des Erys. Diss. Zürich, 1858. Schmidt's Jahrb. 111. p. 310.—*Thoinnet*, Quelques mots sur une variété d'érysipèle traumatique par infection. Thèse de Paris, 1859. Nr. 186.—*v. Bärensprung*, Hautkrankheiten. Erlangen, 1859. p. 70.—*Trousseau*, Clinique européenne, 1859. 26.—1860–1864.—*Hirsch*, Hist.-geograph. Pathologie. Erlangen, 1860–1864.—*Hebra*, Hautkrankh., in Virchow's Handb. der Pathol. u. Therap. 1860.—*Rogez*, Thèse de Paris 1860.—*Campbell de Morgan* (Holmes' System of Surgery), Lond., 1860. I. p. 220.—*Trousseau*, Clinique méd. 1860 (Klin. Vorträge). —*Fenestre*, Sur une épidémie d'érysipèle à forme typhoide, observée à l'hôpital Beaujon. Thèse de Paris 1861.—*Will. Pirrie*, Edinb. Med. Jour., July, 1861.—*Retzius*, Monatsschr. f. Geburtshülfe, März 1861.—*Pihan-Dufeillay*, Union médicale. Août, 1861.—*Feldmann*, Ber. a. Paris. Aerztl. Int.-Bl. 1862. 16.—*Oppolzer*, Allg. Wiener med. Ztg. 1862. 35–37.—*Dannenberger*, Ueb. d. Behdlg. der Rose mit Silbersalpeter. Diss. Giessen, 1862.—*Obé*, Thèse. Paris. (Nr. 174.) 1863.—*Druwith Day*, Medic. Times, 1863, p. 209.—*Th. Billroth*, Langenbeck's Arch. II. 460, IV. 537. IX. 139.—*W. C. Blass*, Diss. inaug. Beobachtungen üb. Erysipelas. Leipz., 1863.—*N. Pirogoff*, Grundz. der allg. Kriegschir. Leipz., 1864. p. 854 ff. 985 ff.—*Larcher*, Arch. gén., 1864. Dcbr. p. 689.—*Jules Simon*, Union méd. 11. Oct. 1864.—*Panthel*, Memorab. 1864.—*John E. Erichsen*, Handb. der Chr., dtsch. von *Thammhayn*. Berl., 1864.—*Béhier*, Conférences de clinique médicale. Paris, 1864.—1865. *Mugnier*, Mag. méd. de la folie consecutive aux maladies aiguës.—*Arlt*, Wien. med. Jahrb. X. p. 147.—*Pujos*, De l'érys. épidémique. Thèse de Paris.—*Malherbe*, Arch. gén. p. 725.—*Charles Martin*, De la contagion dans l'érys. Thèse de Paris.—*Hervieux* (Ueber puerperales Erys.).

Gaz. méd. 1866.—*Durozier*, Gaz. des hôp. 149.—*Anstie*, Lancet. 17. Novbr.—*Eulenburg*, Centrbl. f. d. med. Wissensch. p. 65—*Blockberger*, Quelques considérations sur l'érys. Thèse de Paris.—*Deblieu*, Quelques consid. sur l'étiologie, le traitement et la nature de l'érys. Thèse de Paris.—*Michel*, De l'érys. dans la convalescence à l'asile impériale du Vésinet. Thèse de Paris.—1867. *Daudé*, Traité de l'érys. épidém. Paris.—*B. Withers*, Americ. Journ., p. 280 (Recommending Potas. Iod. internally).—*Liebermeister*, Dtschs. Arch. f. kl. Med. III. 569.—*Löbel*, Ber. d. Rudolphstiftg.—*Bartscher*, Journ. f. Kinderkrankhtn. I.—*Guéneau de Mussy*, Gaz. des hôp. Nr. 10.—*Emil Ponfick*, Dtsch. Kl. Nr. 20 ff.—1868. *R. Volkmann* und *F. Steudener*, Zur patholog. Anatomie des Erysip. Centralb. f. d. med. Wissensch. Nr. 36.—*Volkmann*, Art. Erysipelas. In v. Pitha and Billroth's Handb. der allg. u. spec. Chir.—*Griscom*, Med. and Surg. Rep.—*Mettenheimer*, Dtschs. Arch. f. Kl. Med. p. 203.—*Pozzi*, Gaz. des hôp. 47. —*v. Erlach* u. *Lucke*, Ol. terebinth. Berl. kl. Wchschrft. 44. 45.—*Busch*, Ib. (also 1866).—*Mercier*, Gaz. de Paris 19.—*Trousseau*, Clin. méd. III. éd.—*Pihan-Dufeillay*, Bull. d. Thès. T. 124—57.—1869. *Schwalbe*, Dtsch. Kl. I.—*Labbé*, Gaz. de Paris, 44. 45.—*Colin*, Gaz. hebd. 31.—*Mathis*, Rec. de mém. méd. mil. p. 412.—*Biermann*, Mon.-Bl. f. Augenhlkd. p. 91.—1870. *C. A. Wunderlich*, Das Verhalten der Eigenwärme in Krankheiten. Leipz.—*König*, Arch. d. Hlkde. XI. 23.—*Bayer*, Arch. d. Hlkde. XI.—*Dupres*, Bull. de l'acad. imp. 31. Jul.—*Saklén*, Helsingfors.—*Borgien*, Berl. kl. Wochschrft. 7.—*Heyfelder*, Berl. kl. Wchschrft. 32. 33.—*Gosselin*, Bull. gén. de thérap. p. 289.—*Zuelzer*, Mon. Uebers. in seinem Wochenbl. f. med. Statistik und Epidem.—1871. *Hutchinson*, Ophth. hosp. rep. 32.—*H. Weber*, Med. Chir. Transact., 48, p. 135.—*Haight*, Sitz.-Ber. der K. Acad. der Wissenschftn.—*Bonfigli*, Ippocration 9.—*Wagner*, (Odessa), Mon.-Bl. f. Augenhlkde. X.—*Wilde*, Allg med. Centr. Ztg. (Sulphocarbolate of soda).—*Nyström*, Upsala läk förhandl. 382.—*Westerland*, Finska läk handl.—*Estlander*, Nord. med. Arch. 4.—*Mackenzie*, Brit. Med. Jour., 22, April—*Guéneau de Mussy*, Gaz. des hôp. 104—*Ritzmann*, Berl. kl. Wchschrft. 18.—*Hüter*, Klin. Vortr. 22.—1872. *Ritzmann*, Beitr. zur Aetiol. u. Path. des Erys. Zürich.—*Wilde*, Dtsch. Arch.—*Champouillon*, Rec. de mém. 4411. 3. 320.—*Kaposi*, Arch. f. Derm. u. Syphilis. 36 (Pseud. Erys. in Lup. erytheme). —*Nepveu*, Gaz. méd. de Paris 3.—*Kaczorowski*, Berl. kl. Wchschr. 53.—*Russell* and *Blake*, Brit. Med. Journ., 30. March.—*Dujardin-Beaumetz*, Gaz. des hôp. 51.—*Eulenberg*, his Vierteljschr. Juli. p. 129.—*Heubner*, Jahrb. f. Kinderkrankhtn. 105.—1873. *Holm*, Hospitals tiv. XV. Schmidt's J.—*Wahlberg*, Schmidt's Jahrb. 159 p. 108.—*zum Sande*, Jahrb. f. Kinderkrankhtn. 56 p. 57.—*Savory*, Brit. Med. Journ.—*Squire*, Brit. Med. Journ. Nr. 649.—*Thomas*, Jahrb. f. Kdrkrankh. 144.—*Rigal*, Gaz. des hôp.—*Orth*, Arch. f. exp. Path. u. Pharmak. I. 81.—*Jaccoud*, Gaz. hebd. 25.—*Féréol*, L'Union 36. 41.—*Lordereau*, Journ. de l'anat. et de la physiol. 3 p. 26. Mai et Juin.—*Tutschek*, Bayr. ärztl. Intell.-Bl. XX. 18.—1874. *Sevestre*, Des Manifestations cardiaques dans l'érysipèle de la face. Paris.—*Hüter*. Centralbl. 5. (Subcutaneous Injection of Carbolic Acid). —*Aufrecht*, Ib. 9. The same.

DEFINITION.

Erysipelas is an acute febrile disease characterized by a peculiar inflammation of the skin, which is accompanied by more or less severe general symptoms, headache, loss of appetite, nausea, and sometimes vomiting, and by disturbance of the intellectual functions, with occasional delirium. It begins at the site of some injury to the skin or external mucous membrane, generally suddenly and with chills, and is accompanied by swelling, redness, heat, pain, enlargement of the neighboring lymph-glands, and often by the formation of blisters, and it is disposed to extend rapidly over large portions of the skin. It generally ends in desquamation of the cuticle, speedy resolution, and perfect recovery, prolonged occasionally by the formation of cutaneous abscesses. The only dangers are the inanition which follows long-continued fever in weakly persons, and the secondary inflammations of the lungs, pleura, and peritonæum. Laryngitis and œdema of the glottis may also sometimes bring on a fatal ending.

The duration of the fever corresponds precisely to the continuance of the cutaneous affection, which lasts upon one and the same part from three to four days; if it extends farther, however, and attacks various portions in succession, the duration may reach several weeks. The disease is disposed to attack the same person repeatedly; it is contagious and inoculable; and arises spontaneously under conditions not accurately determined. It occurs, for the most part, sporadically, or as a limited epidemic within the walls of institutions.

HISTORY.

Erysipelas has occurred in a sporadic form, according to the statement of Hirsch,[1] at all times, and, with the exception of the tropics, in all regions of the earth's surface alike.

Its frequency in particular regions may be approximately shown by the following facts: In the whole of England, in the years 1862–1867, erysipelas was recorded as the cause of death in 3,904 children under five years, and in 4,731 older persons (Farr); in Paris there died, on the average, in the years 1865–1869, about 350 persons annually; while according to my own computation some 200 cases received medical treatment in Berlin during six months of the year 1870.

---

[1] Handb. d. histor. geograph. Pathol. I. p. 243.

The disease rarely attains any special frequency, and never prevails in epidemic form amongst an entire community. Many writers have expressed the opinion that it occurs with especial frequency, either in sporadic or epidemic form, as a forerunner or companion of epidemic variola, scarlatina, or diphtheria; but the observations of recent years, which have been rich in epidemics of all sorts, have failed to show such a connection.

Hirsch[1] describes under the names erysipelas typhoides, malignum or gangrænosum, an extensive epidemic which prevailed throughout a great part of North America in the years 1841–1854. This affection, however, as Hirsch himself states, is to be regarded merely as an acute infectious disease closely related to diphtheria, the local symptoms of which, so far as they were manifested in the form of deep-seated inflammation of the subcutaneous cellular tissue, were much more nearly akin to diffuse phlegmonous inflammation (the so-called pseudo-erysipelas) than to true erysipelas, from which it is entirely distinct.

On the other hand, the disease has always prevailed not unfrequently in epidemic form in enclosed establishments, where great numbers of persons live together in hospitals—especially the surgical—in foundling and insane asylums, in military lazarettos and prisons, and in ships. Epidemics in surgical wards have been often described by authors, and Volkmann has collected a great number of them. Every injury, even the slightest, the opening of abscesses, vesication, leech-bites, may serve as the starting-point of erysipelas within the sphere of the epidemic. The following epidemics are likewise deserving of notice:

Rayer describes, according to the statements of Calmeil, an extensive occurrence of erysipelas of this sort in the Paris hospitals, in the year 1828 (especially in those for the insane), where at that time patients were often treated by revulsive applications upon the skin. "For six months," he writes, "the hospitals have been crowded with patients affected with erysipelas. The affection shows itself upon any part of the body, sometimes upon a sound one, but oftener in the neighborhood of some cauterized portion of skin. Similar cases were observed in the Bicêtre, St. Louis, and Charité hospitals, and the number of them at certain times was so great that the disease became a true epidemic. Schönlein mentions in his lectures [1] an extensive epidemic, which he observed in the hospital at Zürich, in 1836. "The affection was characterized in the beginning by traces only of desquamation accompanied by the appearance of pale red spots, which quickly disappeared again. The true erysipelatous redness was not markedly developed until after the use of the cold plunge-bath. At the same time there was considerable disturbance of the sensorium; some lay in sopor; while most were delirious, and did not know where they were; leaving their beds and desiring to go to their work; seating themselves in the middle of the ward to evacuate their bowels, as if they were in the privy."

---

[1] L. c. II. p. 432.

According to Gintrac's description, there was also an extensive epidemic of erysipelas in the hospital St. André, at Bordeaux, in the spring of the years 1844 and 1845. "Both in the private as well as in the surgical wards did every vesication, every incision, every cauterization serve as the starting-point of an attack. Eleven cases on the face and scalp occurred 'spontaneously,' six in consequence of the use of a vesicant; while the same result followed venesection upon the arm." The same hospital was again the scene of a severe attack in 1863, according to Pujos, in which all the cases assumed a typhoidal form. "At this time there was not a single case of pyæmia, while during the previous year eleven fatal cases had occurred."

Trousseau states that in the year 1858 a severe epidemic of puerperal fever prevailed in the Maternité at Paris, and that at the same time a very grave erysipelas was developed among many of the wounded in the surgical division. In recent times these epidemics appear to occur less frequently, although Volkmann observed a severe outbreak of the disease in the surgical clinic at Halle in 1868, where in the summer nearly seventy of the patients in the various wards were attacked by it. In the military hospitals in Berlin, in 1870 and 1871, there occurred 170 cases of erysipelas (1.7 per cent.) among the 9,972 wounded (v. Steinberg). About one per cent. of those sick with typhus were thus affected. More recently, also, reports have been published by Loysel, of an extensive outbreak of erysipelas at Cherbourg, in 1872, and by Savory, at St. Bartholomew's Hospital, London.

ETIOLOGY.

As the term erysipelas is used even at the present day with great diversity of meaning, it is necessary, before proceeding, to define the basis upon which this treatise rests.

Medicine, as distinguished from surgery, has always confined itself, on etiological grounds, to cases described as idiopathic erysipelas, *erysipelas verum s. spontaneum s. exanthematicum* (érysipèle médicale of the French), especially erysipelas of the head and face; while cases occurring among the wounded or after operations have been distinguished from it as *erysipelas nothum s. spurium s. traumaticum* (érysipèle chirurgicale), or e. nosocomiale (Hirsch). The latter term has been used with excessive latitude, so that it is often applied, and especially by English physicians, to phlegmonous and other inflammations of the skin and subcutaneous cellular tissue which tend to spread quickly over large surfaces. Both, as distinctly accepted forms, lead to a very different interpretation.

---

[1] *Schönlein's* klinische Vorträge von Güterbock. Berl. 1842, p. 421.

Opinions about idiopathic erysipelas have diverged widely in three directions since the eighteenth century. . Bromfield, Fr. Hoffmann, Richeraud, and some others attributed it to a status biliosus; Boyer referred its cause to the saburral state; while Lamotte designates as such a stopping up of the pores of the skin which prevents the escape of acrid material separated from the blood.

On the other hand, Richter, of Göttingen, was the first to regard erysipelas as an inflammation of the lymph vessels. Subsequently many physicians were inclined to the theory of a simple inflammatory process; while Borsieri declared it to be an inflammatory condition due to the production of acrid fluids in the body. This theory, in consequence of the authority of Broussais, who looked upon erysipelas as an "inflammation franche et pure," led to the extreme use of antiphlogistic methods of treatment, as with other infectious diseases.

By far the greater number of physicians, however, were disposed to place erysipelas among the acute exanthems.

Sauvages regarded it as an exanthematic inflammatory process produced by corrupt and acrid juices. According to Cullen's view, it arose in consequence of a materia peccans developed within the body, which, in proportion to the amount of fever, was deposited, after the manner of other exanthems, upon the surface. Cazenave, Biett, Schedel, and, later, Rayer, considered it absolutely as an acute exanthem. "It is the exanthematic character of the erysipelatous process," says Canstatt,[1] "which gives it its specific individuality and distinguishes it from other kinds of dermatitis. The true erysipelas develops, like all acute exanthems, from within outwards, in consequence of some peculiar though little known transformative or toxic process in the blood, the local and critical expression of which shows itself mostly upon the outer skin, in the form of an erysipelatous stasis." Schönlein ıtill farther: he was led, in consequence of the resemblance which the appear- ıf the erysipelatous fever bears to urticaria, scarlatina, variola, and so on, to le all these processes in one common family of the erysipelatous diseases.

---

[1] Handb. d. medic. Klinik 1847. II. p. 230.

'hese views, so generally accepted at that time, were first ısed by Trousseau, who regarded erysipelas as the visible ifestation of some intensely active and specific noxious cy working from without, at first locally, like diphtheria, only secondarily infecting the whole organism. The brief ıd of invasion, the frequency of relapse, the peculiarity of its tantly starting from some definite point, and finally the se of the disease, protracted sometimes in wandering erysip- for weeks and even months, afford sufficient characteristic ːs by which it may be distinguished from the infectious dis- ı known as the acute exanthems.

ı accordance with these views Trousseau makes no farther ıction between the so-called idiopathic and the traumatic pelas, inasmuch as he believes that every erysipelas is trau- ;, that is, that it is never developed except as the result of previously existing wound or injury.

ı fact, opinions with regard to the difference between the s of erysipelas described as distinct, rest wholly upon the ıer in which the entrance of the poison is supposed to be ;ed. That the poison in both forms is identical there can be ›ubt; numerous observations, of which several examples are ı below, show that the so-called idiopathic erysipelas occurs, r similar conditions, as well through contagion from the natic form as by its epidemic prevalence in enclosed institu- . The type of the disease itself also, according to Volk- ı, presents in the two forms no remarkable difference, ı it is considered that in the one it mostly attacks persons ced by severe injuries, prolonged suppuration, and the like, ; in the other relatively healthy people are affected. To set- his question, we have only to furnish evidence that in the r, as well as in erysipelas traumaticum, the invasion of the ›id poison depends upon a previously existing injury of the ument, to show that the skin affection is not to be consid- as the result of any preceding general poisoning.

linical observation teaches the strict dependence of the general symptoms upon the local process. The fever runs its course parallel with the steadily progressing or receding inflammation of the skin, and deviates from it in relatively few cases in consequence of severe complications.

Inoculation also shows that the whole morbid process can only be developed by infection through the injured cutaneous covering.

The most various wounds of the skin, and at all periods of their existence, may serve as the starting-point for erysipelas, however, as Volkmann shows in a very comprehensive manner. It is observed as well in severe and fresh wounds after operations and accidental injuries as in unimportant and slight cuts, in trivial fissures and abrasions of the skin, and even in leech-bites; also in suppurations and inflammations of considerable, as well as in those of trifling extent, in wounds in process of granulation as well as in those completely healed.

In cases of so-called idiopathic erysipelas of the trunk and extremities, scarcely an example can be found, according to Volkmann and others, where it has not started from some local disturbance, such as a scratched pustule or the like.

With regard to erysipelas of the face and head, such accordance of observations is, it is true, not obtained. Trousseau found indeed in all his cases slight ulcerations or suppurations of the cavities of the nose or mouth, or in the ear or tear passages, as the starting-point of the process, and many later observations yield the same result; yet in many of these descriptions there remains in this connection a gap, which may in part, it is true, be regarded as the result of defective examination, inasmuch as slight injuries upon the scalp or in the facial cavities may easily escape observation when the parts are highly swollen in the course of the disease. B. König found, for instance, in thirty-three cases of spontaneous erysipelas, a slight injury as the starting-point in nineteen of them, while in the others the examination was hindered by the great swelling of the parts.

The fact that erysipelas begins by preference at the orifices of the body and at the points of junction between the skin and mucous membrane, that is, at points which are most exposed to all kinds of irritation, speaks strongly, too, in favor of the theory. Observation, moreover, shows that the process may be continued from the cutis to the mucous membrane, that is, may affect it secondarily or take just the reverse course.

Positive evidence, too, may be derived in all cases from the so-called habitual facial erysipelas, where the inflammation always attacks the same portion of skin, and generally also starts from the same point; it is impossible to prevent these recurrences until the chronic and generally scrofulous inflammations of the nasal mucous membrane, the lachrymal sac, the ear passages, and so on, from which they start, are healed.

In support of the theory of an idiopathic erysipelas as the result of some general affection, it has also been stated that many acute diseases, especially many that are infectious, show a peculiar disposition to it. Wunderlich, too, distinguishes the following forms of erysipelas: that produced by local disposition; that connected with disturbances of the stomach and intestines; that which slowly wanders; that form, especially the primary and spontaneous, which is analogous to the acute exanthems; that produced by pyæmic infection; the glanders-erysipelas; and finally that form which occurs in the last stages of illness and precedes death only a few days,—forms of erysipelas which, according to his own views, are unmistakably distinct affections, and have little in common besides the form of dermatitis and their name. Many of them should certainly be distinguished as phlebitic, pyæmic, or specific affections of another sort. The classification of the others offers little difficulty, when their etiological conditions are considered. The occurrence of erysipelas as a specific affection in typhus, for instance, can no longer be considered strange, now that Hunter's[1] doctrine, that two so-called specific affections cannot coexist in the same organism, has been refuted.

The complication is, however, by no means so common as to establish a relationship between erysipelas and the specific blood-poisoning.

In the course of typhus exanthematicus, Murchison[2] mentions among 14,476 patients only 92 cases of erysipelas (1 : 159); whilst I have observed it but twice in 229 cases. It may occur at any period of the disease, frequently even in the latest stages of convalescence; it begins generally upon the nose or on one ear, especially if otorrhœa coexists; or starts from abscesses in the eyelids or upon the scalp. In one of my cases the suppurating point of insertion of a Pravaz' syringe was the starting-point; in the other, infected from this, it was the catarrhal affection of the nasal mucous membrane. In typhoid fever it showed itself in about one per cent. of Murchison's cases, mostly the facial form, in connection with otorrhœa. Among 84 cases I have observed it only three times, and Zuccarini eighteen times in 480 patients, especially in those who were suffering from ulceration, suppuration, or hemorrhagic infiltration of the mucous membrane accompanying catarrh of the mouth, the antrum Highmorii, or the cavities of the nose, sphenoid, and middle ear. Zuccarini was able to demonstrate in fatal cases that the dermatitis had developed exactly over those parts most diseased. Among the French, erysipelas as a complication of typhoid fever seems to occur, as in general, disproportionately oftener; at least in six to eight per cent. of the cases of Louis and Chomel. In recurrent typhus Murchison describes only four cases of erysipelas among 1,671 patients. In variola, also, it is not very frequent; in 700 cases, I saw it only seven times, in four of which it started from abscesses and bed-sores, and in the others from markedly suppurating pustules which were deeply seated in the corium. In the single case mentioned by Volkmann also, its starting-point from an excoriated pustule was unmistakable.

---

[1] *Hunter's* Works. Ed. Palmer, 1, p. 313.

[2] Treatise on the Continued Fevers of Great Britain. 2d Ed. Lond., 1873.

When erysipelas occurs in the course of other non-specific diseases, especially in dropsical swellings as the result of renal affections, to which attention has been called by Imbert-Gourbeyre, and in diseases of the heart, its connection with more or less considerable injury is easily established. And in general, persons who are relatively healthy and strong seem to be disproportionately less subject to erysipelas than those who are weak or convalescent from serious diseases, especially such as have been greatly reduced by scrofulous affections, long-continued suppurations, or by syphilis.

The special etiology of erysipelas can be given only within certain limits, on account of the insufficient data at our command.

With regard to *sex*, it is almost universally stated in the text-books that the female is more frequently attacked than the male. This relation, however, does not seem to be everywhere the same; for, in opposition to the French and German reports, Hinckes Bird enumerates, among 260 cases, 147 males and only

113 females. Of the fatal cases in England between the years 1862 and 1868, the male sex numbered 56 per cent. (compare my Wochenblatt für med. Stat. u. Epid. 1870–72).

As to *age*, leaving out of view the earliest days of existence, when the vulnerability of the skin readily conduces to erysipelatous inflammation, it is the prime of life, the period from the twentieth to the forty-fifth year (Volkmann), which is, according to general acceptance, especially concerned. Among 12,556 persons, who died of erysipelas in England in the years 1862–68, 31.1 per cent. were in their first year, 5.9 per cent. were under five, 2.9 per cent. under fifteen, 4.2 per cent. under twenty-five, 12.4 per cent. under forty-five, 20.9 per cent. under sixty-five, about as many under eighty-five, and 1.4 per cent. were still older. It appears, therefore, that the period beyond the fortieth year exhibits the greatest predisposition to the disease.

Any special predisposition connected with definite kinds of business or *occupation* cannot be determined.

As to the *season* of its occurrence, authorities differ widely. Haller found on analysis of the cases which were observed in a period of ten years, in the General Hospital of Vienna, that the majority occurred in April and May, October and November. Chomel and Blache obtained the same results. Lebert[1] states that it occurs most frequently in January and April; while among the fatal cases observed in England, in 1862–67, the greatest number, 30 per cent., occurred in January, February, and March, 25 per cent. in spring and autumn, and 20 per cent. in summer. In Berlin, according to my estimate, the number of cases in 1870 increased from December to May. In the military hospital of that city the majority of Ritzmann's cases (87), occurred in the months of November to January (50), while in the spring and autumn months the disease was less common (6 on an average). Hirsch believes that erysipelas is especially prevalent in times of marked changes in temperature and weather, and in damp weather. The condition of the soil seems to him of less importance, while Naumann is of the opinion that the disease is more frequent upon stony and sandy soils.

---

[1] Handb. d. prakt. Medicin. 1871. II.

Exciting Causes.

Among the most important of the exciting causes of erysipelas must be mentioned its contagiousness. Opinions concerning this point, however, vary widely. English physicians have, since the time of Graves, declared their belief in it; and in France, too, where at the Academy of Medicine it has been the subject of frequent discussions, influential voices have spoken in favor of it; whereas in Germany the question has been but little considered, and only the latest works offer any evidence of its contagiousness

From the not inconsiderable amount of material thus far collected, it follows in support of this view that erysipelas does not occur in the form of extended epidemics, but that, on the contrary, the cases seldom remain single, but often occur one after the other in succession. Starting from the first case, the disease often spreads to those in its immediate presence—nurses, relatives, and physicians—and where, moreover, such infection cannot be regarded as the result of a pervading, local cause. The following instances will serve as evidence of this:

(1.) Wells (1798) describes the following chain of cases. An old man was attacked with erysipelas faciei; one after the other his wife, the housekeeper, their nurse, and his nephew were attacked within a month, all of whom had come in contact with him. (Among the instances of Daudé, Case 4–5.)

(2.) Blackett:[1] After an erysipelas faciei of a patient, his whole family were affected in succession.

(3.) Graves narrates among others a case in which a young man who was taking care of his mother, affected with erysipelas, was himself likewise attacked by the disease. (Daudé, Case 11.)

(4.) Lawrence: A man with a seton in his neck was attacked with erysipelas of the part; his child and wife, who shared the bed with him, were affected, the first with erysipelas cruris, the latter with erysipelas faciei. (Daudé, Case 9.)

(5.) Martin: A fatal case of facial erysipelas in a young woman after abortion. A few days after, the husband, and a lady who had taken care of the deceased, were attacked with the same disease. (Daudé, Case 12.)

(6.) Fenestre, 1861: Puncture of a suppurating bubo and subsequent erysipelas of the part; fourteen days afterwards a neighbor of the patient was attacked with erysipelas faciei, and two days after his death a third man, who had recently entered the hospital, was likewise affected. (Daudé, Case 16.)

---

[1] Lond. Med. and Phys. Jour. Vol. 55.

(7.) Fenestre: Erysipelas in a hospital patient, starting from amputation of the finger; four days afterwards two wounded patients lying by the side of the first were attacked, the one with erysipelas faciei, the other with erysipelas manûs; eight days subsequently a third patient with fistula ani, and nine days later a fourth, a case of cancroid of the nose treated with caustic, were affected, the latter terminating fatally. Both of the last two lay in the same hall and opposite the first case. The almoner of the institution, who had taken care of the original patient, fell sick with erysipelas on the following day and died, and his father, who came to visit him and remained two days with him, was also attacked with it and died. (Daudé, Case 7-9.)

(8.) Gosselin: A rapidly fatal case of erysipelas in a new-born infant. The mother was attacked with the disease, in a scratch upon the leg, two days after its death. (Daudé, Case 21.)

(9.) Daudé: A woman, who was convalescent from erysipelas, visited a female friend suffering from it, and was herself in two days again attacked with it.

(10.) Obé: In Piorry's division of the Hôtel Dieu, two girls, one suffering from odontalgia, the other from angina simplex, were placed in beds near a case of erysipelas faciei. Two days later both were attacked with the same affection. (Daudé, Case 26.)

(11.) Trousseau: A fatal gangrenous erysipelas penis et scroti, following a forcible dilatation of the orifice of the urethra. Twenty-four hours after his death the wife was attacked with erysipelas faciei, following sore throat, and the servant girl, who had taken care of him, was seized with angina and erysipelas palpebr. on the same day.

(12.) Trousseau: Erysipelas mammæ developed in an American lady after the opening of a phlegmonous abscess in the nipple. Her husband, who was absent, returned, and in two days a severe erysipelas developed from a slight scratch upon his leg. Ritchie [1] and others narrate similar cases.

To these and numerous similar cases may be added others, which prove positively that the disease may be directly transported into places which have been previously free from it.

(1.) Bernutz: In November, 1864, a patient with eczema lab. was discharged from the St. Louis, in Paris, because erysipelas had broken out in the hospital. Four days later she was received into the Hôp. de la Pitié, where the disease did not exist, with erysipelas faciei. On the following day her neighbor, who lay opposite, and another patient were both attacked with the same form of the affection. (Daudé, Case 32.)

(2.) In February of the same year an old man with erysipelas entered Gosselin's division of la Pitié. The assistant physician, as well as several patients, were soon attacked with the disease.

---

[1] Brit. Med. Journ., 1871. II.

(3.) Cornil: In January, 1862, a person with erysipelas entered the Hôpital Beaujon. Several days afterwards two patients, convalescent respectively from typhus and variola, and one with pericarditis, were successively attacked with facial erysipelas, and prior to this no case had occurred in the ward. (Daudé, Case 25.)

(4.) Larcher: A patient with erysipelas faciei was the first case of this disease in the Hôp. Cochin, in April, 1864, and he died in a few days. After his death an old man with retentio urinæ, who was placed in the same bed, was attacked with the disease.

(5.) Martin: In February, 1863, a man in the Hôp. de la Pitié, from whose femur a sequestrum had been removed, was, two days subsequently, attacked with erysipelas, for which he was transferred. Three days later three other patients in the same ward were similarly affected. The first man was carried to a ward where erysipelas did not exist, but in three days two other patients, and twelve days subsequently still another were attacked by it; all of them had injuries of various kinds which served as starting-points for the disease.

(6.) Labbé: In December, 1857, an epidemic of erysipelas prevailed in the surgical wards of the Charité. A woman with panaritium was attacked in the part with it, and was transferred to the medical division, in which the disease had not hitherto occurred. She died, and her next neighbor, with acute rheumatism, became affected; and subsequently erysipelas spread throughout this division also.

(7.) Pujos: After a wound in the foot a huntsman was attacked with erysipelas cruris gangrænosum, which proved fatal. His brother, who took care of him, died with e. fac. et cap. His daughter was attacked with e. migrans of the arm and breast, starting from a slight burn upon the hand, and the nurse, with e. faciei. The woman also who washed the patient's linen was affected with an erysipelas of the hand.

(8.) Arlt:[1] A patient with erysipelas faciei was received into the Ophthalmic Division of the General Hospital at Vienna. His neighbor was immediately attacked with the disease; and on the removal of the first case to another ward a still further infection followed.

(9.) Ritzmann: In the Berlin Military Hospital ten cases were observed in 1870–71, infected through other cases, which were in the florid or desquamative stages.

(10.) Broadbent:[2] On the 28th of June, 1871, a man with erysipelas cap. was admitted to St. Mary's Hospital, where there had been no previous cases. A few days subsequently, three cases occurred in the ward in which the patient lay.

In addition to the contagion from patients, erysipelas seems to be capable of spreading from the dead body; at least two cases have been observed by Maclagan[1] and Rogez (1860) in which those who made the post-mortem examination of patients dying of erysipelas were affected with it, although their hands showed no injury, and after a certain period of incubation, so that direct inoculation could be excluded.

---

[1] Oesterr. Med. Jahrb. 11, p. 147.

[2] Brit. Med. J., 1871, 2, 94.

With regard to the *way* in which erysipelas is transferred from a patient to a healthy person, no direct observations exist. The question also as to the *stage* in which the disease is most infectious has not yet been solved, although some instances show that it is contagious as well in the florid as in the desquamative period. The action of the poison does not seem to extend over great distances, since any great number of healthy persons are never attacked simultaneously from one patient, as in typhus, for instance, but the disease progresses successively n hospitals from one bed to another. In the Berlin Military Hospital, according to Ritzmann, it travelled in six cases directly from one bed to the next; and Savory observed, during a great epidemic in St. Bartholomew's Hospital, in 1872, that the disease almost always attacked the patient lying next the one previously affected.

Campbell mentions a very remarkable case communicated to him verbally by Goodfellow, which I quote from Volkmann: "In the course of an epidemic of typhus, which prevailed in the fall of 1833, erysipelas also constantly showed itself n the fever hospital, spreading, as a rule, from bed to bed. The most striking example of this propagation was in a large ward containing seven beds upon one side and six upon the other. The patient lying in the second bed upon the former side was attacked with erysipelas. Then the patients in bed No. 3, and in No. 1, were simultaneously affected, and afterwards the one in No. 4, and subsequently all the patients were attacked in regular order upon that side up to and including No. 7. From this point the erysipelas extended to the opposite side of the ward, beginning with the bed at the same end as No. 7, and wandering back from bed to bed without missing a single one, until No. 12 was reached. The patient in bed No. 13 was alone spared.

The poison of the disease offers considerable resistance to the action of disinfectants, and appears capable of remaining active also for a long time. In several epidemics, for instance, in the year 1822, in the infirmary at Montrose, which Gibson observed, they were obliged to evacuate the affected ward, as no means availed to check the spread of the erysipelas when introduced. At the present day also it is shown by many epidemics (one in the surgical clinic at Halle, in 1868, described by Volkmann, in the Paris hospitals, in the Berlin Military Hospital, in St. Bartholomew's Hospital at London, and others), that the most careful and thorough measures do not suffice to control the spread of the disease where it is impossible to immediately isolate those first attacked.

---

[1] Edinb. Med. Journ., 1837.

The poison of erysipelas, which, according to the examples quoted, effects its spread by contagion, that is, diffuses itself through the air to a certain distance, is also to a decided extent *inoculable.* The serous fluid of the vesicles and of the œdema of the skin and subcutaneous cellular tissue seem to be especially infectious by direct transfer. The earlier attempts by Willan, who inoculated with the fluid contents from the vesicles of erysipelas, and produced only a red and painful (but not spreading) inflammation of the skin similar to erysipelas, as well as those of Martin, from which only a temporary inflammation of the inoculated places resulted, are to be regarded as negative evidence of little account. It has not unfrequently happened also that vaccine matter has been taken from children who immediately afterwards have been affected with erysipelas, and used without bad result.

On the other hand, Doepp[1] communicates a very remarkable case of a positive character. Lymph was taken from a child that was attacked the day afterwards by vaccinal erysipelas, and nine other children were vaccinated with it. All of them had erysipelas.

The disease has also been frequently communicated by dressing wounds with the aid of instruments which had previously been employed for the same purpose in erysipelas. Ritzmann shows the probability of such an origin in many cases which have occurred in the Berlin barracks, and, with many other surgeons, laments the powerlessness of the measures employed against a direct transference, a colportation, of the infecting matters by means of instruments, lint, and other dressings.

---

[1] Mitthlg. aus d. Arch. d. Ges. corr. Aerzte in Petersb. 1840. *Schmidt's* J. 30, p. 184.

Direct transference is also positively proved by experiments upon animals. Some of the earlier attempts, like those of Ponfick, were only partially conclusive; but lately Orth has succeeded in demonstrating, in a series of twenty-three experiments, that erysipelas can be successfully inoculated from man to the lower animals.

The fresh contents of the bullæ of erysipelas bullosum in man, the expressed œdematous fluid from the portions of skin affected with erysipelas in the rabbits used for experimentation, as well as the pus formed at the point of inoculation, and the blood of the affected animals filtered through linen, were found to be actively contagious. If any of these fluids became putrid, their action was somewhat retarded, but did not fail. An artificial infecting fluid also, which was obtained by the cultivation of the serum, containing abundance of bacteria from the bullæ, in Pasteur's solution (10 per cent. sugar, 0.5 per cent. phosphate of ammonia), was found to be effective.

As the most characteristic appearances of successful inoculation, Orth noticed an affection of the skin, accompanied by a rapidly increasing fever, beginning in small spots, with œdematous infiltration and sharply defined redness, and once even with the formation of bullæ, which extended with more or less rapidity. The parts first reddened became paler after a time, and generally assumed a more yellowish hue, or in the most acute forms of infection, a green, gangrenous color. In the affected parts various large infiltrations of the skin and subcutaneous cellular tissue were found, the young cells, of which they consisted, forming abscesses in the deeper layers, where they were most abundant, but less frequently in the upper layers. This form of disease, which may farther be produced by inoculation from infected to healthy animals, differs manifestly from the phlegmonous and rapidly suppurating dermatitis in putrid poisoning; there a minimum amount of the infecting fluids (1–2 cc.) sufficed, while for this poisoning relatively larger quantities are necessary; moreover, in contrast with putrid poisoning, the artificially cultivated fluids were effective.[1]

---

[1] *Lukomsky's* essay (*Virchow's* Archiv, 60, 3. and 4. Heft) may also be referred to, confirming the truth of these statements. His observations are also important in this

I deem these experiments of Orth's as conclusive, moreover, because they produced appearances identical with erysipelas occurring spontaneously upon domestic animals, swine, horses, sheep, and the like.[1] At all events, they serve to confirm clinical experience concerning the possibility of successfully inoculating erysipelas.

From the observations above quoted, it seems very probable that a great number of the cases of erysipelas arise from contagion, or by direct inoculation.

The isolated, single cases, however, which occur among people without any connection with one another, and those cases which in one and the same locality appear in multitudes and simultaneously, can only be referred to an autochthonic origin. The following instances illustrate the affection as it occurs in this rare manner:

(1.) Trousseau observed in a private house a regular epidemic, which affected simultaneously nearly every tenant, even to the concierge. Direct transference of the disease seems scarcely probable in such cases.

(2.) Daudé relates the following instance: At the end of November, 1864, a merchant in Paris was attacked with facial erysipelas, and was taken care of by his wife and maid-servant. The cook did not enter his room, but eight days after his recovery was attacked with the same form of the disease. On the same day two operatives, who had no connection with the first patient, were affected with the disease. Twenty-one persons, of whom eighteen were girls or women, were attacked one after the other. At the same time numerous other cases occurred in the city and environs.

Some authors would refer the sporadic cases to a "pyæmic infection," the relationship of which to erysipelas is maintained on various grounds, especially from a surgical point of view. Roser, for example, regarded many cases of erysipelas occurring in hospitals as the expression of a mild miasmatic pyæmia, and Pirogoff could not conceive of erysipelas without pyæmia, or of the latter without erysipelas. Not to mention the fact that the latter expresses himself like Volkmann, who declares against

---

respect, that it is not merely subcutaneous injections, but also the mere painting of the fluids, taken from parts affected with erysipelas, upon wounds, which will produce a similar inflammation.

[1] See *Spinola*, Path. u. Ther. für Thierärzte. Berl., 1863. p. 28.

such a connection, and extends the conception of "traumatic erysipelas," probably after the custom of many English physicians, to phlegmonous inflammations as well, numerous observations do not show any such relationship. In the epidemic at Bordeaux, for instance, where, according to Pujos, thirty-four per cent. of those affected died, no case of pyæmia occurred. In Volkmann's Military Hospital at Trautenau (1866) there were pyæmic affections, but no erysipelas; and Ritzmann shows that in the Berlin Military Hospital, in 1870, pyæmia occurred during the first months of its occupancy with great frequency, while the cases of erysipelas exhibited a frequency very different from it in point of time as well as duration.

Hitherto no one has succeeded in discovering the noxious influences which lead to the spontaneous development of erysipelas in certain localities. There has been a disposition to connect it with the *crowding together of many persons in confined institutions*, but this opinion is not sustained by experience; for the disease, which it is true never dies out in many old and unwholesome hospitals, often occurs very extensively in the most modern establishments, which fulfil every claim of cleanliness. Erysipelas is indeed relatively rare in prisons, transport-ships, and even in military hospitals, where great numbers of severely wounded, with suppurating wounds, lie crowded together under the most unfavorable circumstances. In the Crimean war, for instance, while pyæmia and hospital gangrene were claiming numerous victims, it was scarcely ever observed by the English; and in the war of 1866, Volkmann did not see a single well-marked case in Trautenau and other hospitals, where about 1,000 wounded were quartered. During the war of 1870–71 it occurred very infrequently, according to my observations, in some of the lazarettos established in France, but in the well-situated military hospitals in Berlin, on the other hand, it attacked two per cent. of the wounded (v. Steinberg).

In other cases the cause of the disease has been sought in the pollution of the soil, as in the frequent overflowing of the cellars of the Hôtel Dieu by the Seine, for instance; and yet this theory is unsupported by any considerable number of observations.

Several instances seem to me nevertheless to point to more definite local causes. Volkmann's treatise narrates that in the Middlesex Hospital, for a long time, those patients exclusively who lay in two beds, standing next each other, were constantly attacked one after the other with erysipelas, without any farther infection occurring in the same ward. It was finally discovered that a pipe from the privy, which was situated in the wall between these beds, was defective; it was repaired, and the erysipelas disappeared. Ten years afterwards erysipelas began again in the same beds; and, mindful of former experience, the pipe was examined, and again found defective; on mending it no new case occurred. An entirely similar instance was observed in the surgical division of the Berlin Charité, where the disease occurred only in certain beds which stood directly above a defective privy pipe; after repairing it the erysipelas disappeared.

To these cases may be added a very important observation by König. During a slight epidemic, at the clinic at Rostock, it was noticed that only wounds from recent operations, and indeed upon such patients only as had been operated upon in the operating theatre, were attacked with erysipelas. The cause of this infection was finally suspected to be the use of the pillows of the operating table, which were saturated with blood; and in fact after their removal the erysipelas disappeared. This theory received farther support by the following experiment: The pillows were extracted with water for twelve hours, and with this rabbits were inoculated. One attempt was without result; in the other there was developed from the point of inoculation upon the back a diffuse dermatitis, which spread over the belly, produced œdema of the præputium, bullæ and crusts upon certain places, and disappeared after twelve days.

These observations are not yet numerous enough to afford a basis for positive judgment concerning the noxious influences which give rise to autochthonous erysipelas; they may, however, be regarded as an important starting-point for farther investigation.

PATHOLOGY.

Symptoms.—Erysipelas generally begins suddenly with chills, less frequently there may be a feverish condition for a day or two beforehand.

The first local symptom is a feeling of warmth, tension, and pain in the skin, which soon shows œdematous swelling, and becomes red. The redness is at first pale and in spots, it then becomes diffused and more intense, and disappears on pressure with the finger, to return again when this is removed. The swelling reaches a high degree early, especially in places where the skin is attached to the underlying parts merely by loose cellular tissue. On account of the great stretching which it undergoes, the skin appears smooth and shining. At the same time the painfulness of the affected part increases, and it is intensified by pressure, which leaves a pit in the swollen skin. The lymph-glands in the neighborhood swell more or less, but return to their natural size immediately on the disappearance of the cutaneous inflammation.

Most frequently it is the skin of the face and head which is affected, less often that of the extremities and trunk; and slight or more severe injuries of the openings leading to the cavities of the body, of the nostrils, eyelids or meatus of the ear, or of the mucous membrane of the nostrils or throat, serve as the starting-point.

The redness and swelling reach their highest degree of development on the second or third day, and in facial erysipelas patients at this time are greatly disfigured, and are scarcely to be recognized; they can with difficulty open the œdematous and prominent eyelids. On the third or fourth day the parts first affected become gradually paler, the swelling and painfulness diminish, the skin becomes wrinkled, desquamates in branny scales or larger flakes, and only retains a slight yellowish or brownish color which soon disappears.

The *progressive course* of the skin affection is especially characteristic of the disease. While the erysipelatous inflammation is still in the stage of efflorescence, or already subsiding at the part first affected, it spreads at uncertain intervals to the neighboring parts, and there reaches its height at correspondingly later periods. On the side where it is advancing, the redness which elsewhere runs gradually into the color of the neighboring skin, is generally marked by a sharply defined and often wall-like elevated edge. The course of its extension is often preceded by peculiar tongue-shaped projections beneath the cutis.

Erysipelas faciei et capit. is limited mostly to a more or less extensive portion of the skin of the head and face, and seldom descends over the neck to the trunk. Upon the latter, and upon the extremities, it often wanders over large surfaces. The course of the disease is, therefore, in the first case, only three or four days, or one, two or more, seldom three weeks; but in the latter situations it is often much longer, and in some cases it reaches a duration of several months.

In proportion as the œdematous swelling of the skin advances, and to the more or less acute progress of the disease, the epidermis is often raised in smaller or larger bullæ, which are filled with a serous fluid of a yellowish or bloody tint. These subsequently burst and dry up, or form crusts.

Simultaneously with the cutaneous inflammation, the temperature and the pulse-rate rise rapidly and considerably, the former even to 41° C. [= 105.8° F.] and upwards. The patients complain of headache, prostration, loss of appetite, and often of nausea, vomiting, and great thirst. The tongue is thickly coated, and, when the fever is high, dry; the hepatic region is often painful to pressure; and the urine scanty, turbid, and often albuminous. The sleep is uneasy, disturbed by wild dreams, and in erysipelas of the head and face often wholly absent. Not unfrequently delirium is present.

The mucous membranes bordering upon the affected skin—those of the nose or throat, of the eyes, vagina, etc.—are almost always, and often in a high degree, affected secondarily by the inflammatory process, which also is primarily developed upon those superficially situated, and may, by extension to deeper parts, especially in the air-passages, give rise to grave disturbances. Erysipelatous angina renders swallowing difficult, and by extension not infrequently leads to laryngitis, bronchitis, and even pneumonia.

The termination is almost always in recovery with persons previously healthy and strong. The fever, which runs parallel with the cutaneous inflammation, ends simultaneously with it; the other symptoms quickly abate; only convalescence is occasionly disturbed by numerous abscesses, which often appear for weeks after the cessation of the fever. The patients exhibit a strong disposition to relapses, which sometimes return more or less periodically.

Anatomical Changes.

Inasmuch as erysipelas is seldom fatal or accompanied by severe chronic affections, the number of post-mortem examinations in uncomplicated cases is very small; most of them concern cases which have been complicated by other affections, and where death has been caused by the latter. On this account our knowledge of the anatomical changes peculiar to erysipelas is but little advanced, and the various statements which have been collected, by Daudé and Ponfick for example, differ widely from one another. Excluding the conditions which belong to the complicating affections, of most diverse character, the following may with probability be referred to erysipelas.

Inflammation of the *mucous membrane* of the throat is often met with in facial erysipelas; with this is connected, at one time, inflammation of the mucous membrane of the nasal, frontal, and Highmorian cavities (Zuccarini), at another time, of the larynx (often in such cases with severe œdematous swelling), and of the bronchi and the lungs. (Cases by Wutzer, Avery, Hebra, Fenestre, Labbé, Peter, Trousseau.)

Independently of this, *hypostatic congestion* and *croupous pneumonia* are found at times (Thoinnet, Trousseau, Ritzmann, and others). Serous and purulent pleurisy is described by Lawrence, Velpeau, Daudé, Ritzmann, and others, and anterior mediastinitis by Lawrence and Ponfick.

Enteritis in the lower portion of the duodenum was found by Lawrence, Hebra, Bennett, and others; considerable hyperæmia, with intestinal hemorrhage, by Bayer; small duodenal ulcers in the neighborhood of the ductus choledochus, by Malherbe and

Larcher; and peritonitis by Trousseau, Hebra, Wilks, Ritzmann, and others.

The *spleen* is unanimously described as enlarged, softened, and often liquefied (Ponfick), while with regard to the condition of the liver the statements are entirely at variance.

The *kidneys* were always found by Ponfick in a state of parenchymatous nephritis.

Heart.—The statements of Sevestre concerning the frequent occurrence of endocarditis and pericarditis have not been sustained by dissections, the latter only having been found twice in the dead body.

Meningitic affections seem to occur only as the result of wounds or other complications.

The blood was found by Hebra, when death occurred at the height of the disease, bright red and thin, but when the patient died after any considerable exudation, thickened; by Pihan-Dufeillay it was found dark-colored and thin; by Ponfick, dark, thin, and varnish-like. According to Virchow, the proportion of fibrine is increased.

In contrast with these meagre and dissimilar statements, there appears as a uniform condition, according to Ponfick's observations of eleven cases, a more or less marked parenchymatous degeneration of the muscular tissue of the heart and vessels, as well as of the extremities and trunk, and of the liver and kidneys; also softening and hyperplasy of the spleen.

The affection of the skin has been studied more thoroughly.

According to Biesiadecki's[1] beautiful investigations of the facial form of the disease, which agree with those of Volkmann and Steudener, simple erysipelas, like phlegmonous inflammation, affects the skin in its whole thickness and the subcutaneous cellular tissue.

The rete Malpighii is dried up in the dead body almost to a horny layer or crust, and when removed there remains attached to the swollen papillæ only the deepest row of cells, which are loosened in their connection, and, like the epidermal cells of hair follicles and the outer layer of the sebaceous glands, are much swollen.

---

[1] Sitzungsber. d. K. Acad. der Wissensch. zu Wien. 1867. II. p. 231.

All the layers of the corium and of the subcutaneous cellular tissue are œdematous, swollen, and penetrated by large, finely granulated, white blood-corpuscles. The meshes of the connective tissue in which these cells are embedded are very decidedly separated by them and by the fluid which uniformly permeates the tissues. In proportion as the cells are massed together in quantity, the individual fibrillæ lose their sharp outline, become broader, swell up, and finally leave behind only a homogeneous matter, which is faintly colored by carmine. In this way an abscess is formed, situated most commonly at the tips of the papillæ, and traversed only by solitary and often torn elastic fibres.

The blood-vessels are enlarged, and filled with corpuscles; their walls, however, are unchanged, and sharply defined against the surrounding masses of cells. Only in the proximity of the abscess formation does their contour disappear, and beyond this their course is marked only by a brown mass of decomposed blood. As soon as the skin becomes pale, however, after the second or third day, according to Volkmann and Steudener, a rapid destruction of the escaped elements begins. In the subcutaneous cellular tissue there are very soon to be found only masses of cells in a state of finely granulated disintegration, and a few hours afterwards often only granular detritus. In the upper layer of the cutis only the lymph vessels are thickly filled with the uniform granulated cells or surrounded with large collections of them. A portion of them are, perhaps, taken up by them; but the most of them, however, rapidly perish, for in a day or two afterwards there is often not a trace of the affection to be found in the skin.

We now come to the above-mentioned numerous observations upon the extensive occurrence of globular bacteria (punctiform bacteria, Ehbg.; microspheres, Cohn) without spontaneous movement. They were first discovered by Hüter, then by Nepveu, Wilde, Orth, and Wahlberg, everywhere in the inflamed tissues, and especially abundant in the œdematous parts, much less so in the blood.

On reviewing these results, we find little that is specifically characteristic. The parenchymatous changes of the large glands and of the heart have been recognized, since Liebermeister's observations, as conditions which are common to every sort of severe febrile affection. The intense cutaneous inflammation, with the abundant œdema, occurs also in phlegmon. The slight differences consist only in the relative integrity of the peculiar parenchymatous tissue, in the absence of destruction of the fat cells, of the liquefaction of the intercellular substance, and in the rapid restoration of the normal condition in erysipelas (Volkmann). The bacteria also, which are described, offer no morphological peculiarities.

The anatomical changes, therefore, will not suffice to answer the often proposed question, whether erysipelas constitutes an independent disease, or is only a simple inflammation of the skin without specific character, as Hebra and lately Klebs (Handb. der pathol. Anat.) regard it. In fact, Virchow,[1] Heyfelder, and others are of the opinion, that in addition to the infectious form there may be a non-infecting erysipelas, produced by thermal, chemical, or mechanical influences, which possibly may become infective secondarily, and then for the first time assume a specific character.

In considering the anatomical changes, we must also at the same time have regard to the course of the affection. The most important distinctive feature in erysipelas, besides its fugacity, is its disposition to spread only by creeping uninterruptedly onwards without making jumps. This peculiarity finds its anatomical expression, if I may so speak, on the one hand in the direct continuation of the inflammatory process to mucous membranes which are in direct connection with the affected portions of skin, on the other hand in the absence of more grave changes, especially those of a pyæmic character, as large abscesses at the height of the disease, embolus and infarction, diffuse inflammation of serous and synovial membranes, and the intestinal affections so common in septic poisoning. (That pyæmia and erysipelas do not exclude the coexistence of each other, is shown by some observations of Ritzmann, in which the latter was complicated with pre-existing pyæmia.)

---

[1] Ueber Lazarette und Baracken. Berlin, 1871.

In comparison with this, no inflammation of the skin produced by external thermal or other irritants shows this peculiar method of spreading. A more or less extensive, diffused phlegmon and lymphangitis, such as many observers at least have in view, does not run its course without leaving behind it corresponding local changes.

But is the exciting cause, the morbid poison of erysipelas, that always runs a uniform course, varying only in intensity and extent, is it constantly the same, or may, perhaps, products of decomposition of various kinds, or animal poisons, produce the same affection? Billroth admits the latter theory, although he has abandoned his former distinction between an erysipelas which is produced by self-infection in consequence of secretions retained and decomposed in wounds, and one which is the result of infection from without. Observations in the French war, where great numbers of wounded were crowded together, showed him that erysipelas was developed under such circumstances less frequently than was to be expected according to such theory. The question may be considered as settled up to a certain point by the inoculations which have been made. The specific poison always yielded (Orth, Lukomsky, and, apparently also, König) a well-marked erysipelatous inflammation, even when only small quantities were used. Putrid infection, however, which, according to my own and numerous other experiments, requires relatively large quantities of the infecting material, produces sometimes extensive suppurations and abscesses of the cellular tissue, but (with the single exception of a case by Ravitsch) no affection resembling erysipelas. (From the additional experiments which Orth has in view, we may expect further advances in our knowledge of this subject.)

In what substance we are to seek the morbid poison has not yet been positively determined, but a contemplation of the observations in hand leads to several established points.

We have to consider a disease which is inoculable and contagious, and may under given circumstances exist spontaneously, as well as a fixed poison which can also diffuse itself through the air. The affection starts from some solution of continuity of the skin or superficial mucous membrane; upon more deeply situated parts, the stomach or lungs for example, it never arises primarily. As soon as the irritating action of the poison begins, the inflammation of the skin spreads outwards, step by step, from the affected part, halting, according to Pfleger's investigations, at places where mechanical obstacles or the resistance of pressure stand in its way, or turning aside for them, and returning again, indeed, to the part previously affected, without leaving behind any serious disturbances even after frequent relapses. This process can with difficulty be explained otherwise than as produced by an irritation which is always present within the sphere of the existing inflammation, and which is constantly being pushed forwards mechanically. Every fluid would behave under the same conditions of pressure just as does the œdema, which plainly carries the irritant along with it. The lymph-vessels, also, which have often been regarded as the exclusive channels for the distribution of the poison, are affected, as will be shown farther on, only in the same degree as the other tissues within the area of the inflammation.

Matter chemically different, such as the products of decomposition of the albuminates, could not give rise to such a condition, even were it considerably increased, since it would leave behind it, at the part first affected, much more serious after-effects than erysipelas, which, in spreading, leaves the part first involved already in process of recovery. At all events, under this supposition it could not be explained why the local affection limits itself to the skin, and why in its spread it follows purely mechanical laws.

We can think rather only of an agent which is in the broadest sense ferment-like or mechanical in its action, with an activity and vitality of short duration only, but with great capability of reproduction. In the present state of our knowledge we are led to attach ourselves to the indications presented by the bacteria, which as carriers, or exciters, or at least as attendants of the process, are surely found. In addition to this result, the published investigations of Lukomsky really point to the same conclusion. It appears by these that in the animals experimented on, the bacteria (micrococci) were found only luring the existence of the dermatitis, and that they disappeared simultaneously with its expiration. The products of the local irritation, especially the cellular infiltration, disappear in the same way, or at least only a little more slowly, according to the above-mentioned investigations of Volkmann and Steudener. While by this coincidence a connection of the affection with these organisms is made probable, some experiments of Orth's show, moreover, that the œdema of erysipelas is at least weakened in its infectious properties as soon as the vitality of the bacteria is impaired by the use of carbolic acid and the like.

After this we are no longer authorized in rejecting the theory which regards these objects as at least local exciters of disease. The strict dependence of the general symptoms upon the cutaneous inflammation is, moreover, very remarkable. Even the increase and diminution in size of the spleen proceeds mostly parallel with it. A beautiful proof of this was furnished by Friedreich, among others (Samml. klin. Vortr. 75), in the case of a patient with a wandering spleen, in whom, by means of palpation, its enlargement and diminution, synchronous with he course of erysipelas, could be demonstrated. Can any irriant be conceived of, in our present state of knowledge, the eneral and local action of which begins and ends simultaneously, unless it is sought in the vital process of these organisms?

To be sure, it cannot yet be determined in what way the morbid symptoms are brought about. It is regarded as possible hat bacteria may produce a matter which may act as an irriant both locally and generally; but such an one would, if soluble, hardly confine itself within bounds limited purely by mechanical relations; otherwise, however, the irritation could not immediately follow the advance of the œdema.

It may be conceived, therefore, that these organisms may produce a mechanical excitement of the cutaneous nerves by heir rapid increase alone. Such irritations may, however, as is well known, in addition to the local symptoms, such for instance s follow the use of epispastics,[1] produce, according to their trength, a more or less prolonged and considerable increase of temperature, and a corresponding effect upon the vascular system. In favor of this, on the one side, are the observations of Naumann, ; on the other side the well known works of Liebermeister,[1] Gildemeister,[2] and others, and the investigations, moreover, of Kuntz and Röhrig,[4] Paalzow,[5] and Haidenhain (Pflüger's Arch., III. 504), which show that a fall of temperature takes place within the body by irritation of the sensitive nerves, cannot be regarded as standing in opposition to this. These investigators were concerned with different periods in the course of the experiment, which, as Virchow suggests (in his Arch. 52), may possibly bear the same relation to each other as the narrowing of the vessels which accompanies irritation does to the hyperæmia which follows it.

---

[1] *Zuelzer*, Deutsche Klinik 1865.

This view is supported, too, by the evidence of increased consumption of oxygen and production of carbonic acid, as well as by the increase in the excretion of urea, although small, as determined by Beneke[6] and others.

It must, it is true, be granted that the increase in temperature and metamorphosis of tissue falls far short of the rise which takes place in fever. It is easily conceivable, however, that an excitement of the cutaneous nerves of a more appropriate sort than the electric point or a cold bath affords, aided by the rapid emigration of the white blood-corpuscles, might give rise to much more intense symptoms.

More positive data in these directions, however, can be obtained only by experimentation. Many an experiment in therapeutics has been based upon a similar supposition, although the result does fall short of the expectation. If, then, our knowledge of the nature of this mysterious disease is enriched by the proof that it is connected with the immigration of these parasitic organisms, only by a fact which requires for its full valuation further investigations in a new direction, it is still a great gain that a departure may be taken from a definite point.

---

[1] Prag. Vierteljahrsschr, 1865. *Pflüger's* Arch. 1872. p. 196.

[2] Prag. Vjahrsschr. 1864. Arch. f. klin. Med. X. ff., *Virch.* Arch. 52 ff.

[3] Ueber die Kohlensäurenproduction bei der Anwendung von kalten Bädern, etc. Basel, 1870.

[4] *Pflüger's* Arch. 1871.

[5] Ib. 492. 1871.

[6] Grundlinien der Pathologie des Stoffwechsels. Berlin, 1874. p. 117.

Symptomatology.

Integument.—The local affection begins generally with swelling and painfulness of a small portion of skin, which soon becomes rose red, and later darker in color, and rapidly increases in size. New spots appear near it, which become confluent, and in a few hours, according to the locality affected, the skin, of the size of a dollar or the palm of the hand, is colored deep red, swollen, and hot, and the patient experiences a stinging or burning pain in it. On slight pressure with the finger the redness can be made to disappear temporarily, and a pit is left in the distended skin, which gradually fills up again. On puncture a serous fluid mixed with blood is discharged. Pressure produces also a lively pain.

Erysipelas has the disposition, moreover, to spread superficially with greater or less rapidity (erysipelas ambulans s. serpens). It seldom remains confined to a small area (e. fixum), but often spreads from the part first attacked over wide regions of the skin, from the head at times to the neck, from there to the back and front of the trunk, to the upper or lower extremities, or follows the opposite course, according to its starting-point. Upon the extremities it often attacks large portions of the skin.

Until very recently no explanation of the apparent irregularity in the method of its extension had been discovered. The early theory of Billroth's, that the erysipelas really followed the course of the lymph vessels, had not been confirmed. Only lately has Pfleger, at Billroth's suggestion, collected observations upon this point in fifty-five cases. His results are therefore of special importance to our comprehension of the course of the disease, as they show that its extension is determined in accordance with the above expressed opinion solely by the mechanical relations of the pressure exerted through the cutaneous coverings.

The advance of the erysipelatous redness in the skin takes place in many cases in the same way, to use a striking illustration of Billroth's, as water spreads in blotting-paper. The redness and swelling of the skin is not sharply defined, but gradually passes into the normal skin; only on one side the border is generally formed by an abrupt, wall-like, and somewhat elevated edge (e. marginatum), and it is from this that the extension mostly proceeds, rounded, tongue-like, subcutaneous projections shooting out, which are followed by a broader advance. The appearances are generally complicated, more particularly upon the anterior and inner side of the thigh and the external surface of the thorax, by a redness of the subcutaneous veins and the lymph vessels, extending from the border of the erysipelas to the nearest lymph gland. While, however, the skin is movable over these, the points or outshoots, according to Pfleger's investigations, lie apparently nearer the upper surface of the cutis. Their extension seems to depend solely upon the architectural relations of the cutis over the various parts of the body.

The investigations of Langer[1], Biesiadecki, and others show, as is well known, that the main lines of the bundles of fibrous tissue in the subcutaneous tissue cross each other in rhombic meshes. If holes are made with a round punch in the skin, they are drawn out into slits according to the direction of the longitudinal diagonal of these rhombic meshes, which also determine the arrangement of the meshes of the networks of the blood and lymph vessels, and which designate the direction of the tension to which the skin is subject. Over all regions of the body where, in general, tension exists in an upright symmetrical attitude, and especially where it is not uniform, a great stretching constantly takes place in the direction of these slits. In their direction, that is, in that of the greater tension, the spread of the erysipelas meets with less resistance than in any other. The projections, also, which accompany the extension of the erysipelas, push themselves forward according to the lines of these slits. Inasmuch, now, as the blood and lymph vessel districts, especially on the extremities, correspond to these, the serous exudation is also pushed forwards in these directions in consequence of the less resistance.

In places where the skin is of thicker structure and more firmly attached to its base, by means of the so-called ligamentum cutis around the base of the skull, at the condyles of the joints, over the crest of the ilium, Poupart's ligament, the spinal tibiæ, and so forth, and at places, too, where two portions of skin possessing a different cleavage border upon one another without crossing, a real hindrance to the farther extension of the erysipelas seems to be offered. It progresses with striking slowness in these regions, halts, or goes around them and leaves them untouched throughout its whole course. Thus, for example, erysipelas, when it starts from the mamma, remains stationary at a horizontal boundary line on a level with the xyphoid process. It confines itself, moreover, generally to the face and head, and moves, when it progresses, only over the scalp towards the neck, and from thence onwards. The same method s followed when the direction pursued is reversed. In this ase it spreads from the scalp to the ears, thence to the forehead, heeks, eyelids, nose, and finally to the lips. The chin remains lmost always free, even when the erysipelas starts upon the ead, at the nose, or at the corner of an eye for instance, probbly because a perpendicular tension prevails in the skin of the hin, while immediately in the neighborhood it ascends towards he angle of the mouth obliquely, and extends into the region of he cheeks in a horizontal direction.

---

[1] Zur Anatomie und Physiologie der Haut, (1) über die Spaltbarkeit der Cutis, (2) über die Spannung der Cutis. Sitzungsber. der k. Acad. d. Wissensch. z. Wien 1861.

The arrangement of the papillæ in the papillary bodies in ows corresponding to the folds or furrows in the skin also serves s an obstacle to the direct spread of the erysipelas. It evades he folds, the plica naso-labialis, the furrows in the forehead nd buttocks for instance, inasmuch as it pushes itself forwards ot continuously, but from their terminal angles.

In portions of the skin which are firmly bound down to the ubcutaneous base the formation of the outshoots fails more or ess, and the progress at such parts takes place mostly in the orm of a broader advance.

The unequal rapidity with which erysipelas extends over ifferent parts of the skin also seems to be in conformity with hese observations. It spreads most quickly upon the back and calp, upon the shoulder in the direction of the outshoots from ehind forwards, and upon the upper arm and thigh centripetally. Over the rest of the body Pfleger noticed no special difference in the rate of its progress; but it appears to spread everywhere more quickly in the direction of the points than perpendicularly to them. It extends as well centripetally as centrifugally, probably because the œdema which distends the cutaneous tissues to a considerable extent is constantly pushed forwards, when greatly increased, towards the point of least resistance, that is, parallel with the direction of the cleavage of increased tension and of least pressure.

Some descriptions mention cases where island-shaped portions of the skin, not in direct connection with the main disease, are affected, erysipelas erraticum. In fact, as Pfleger observes, erysipelatous patches several centm. distant from the borders of the dermatitis not unfrequently occur. In these cases, however, the intermediate skin is always painful at some points; the process being continuous, therefore, although in the interval it has not attained a full development. There is often found, also, a zone of a certain size beyond the reddened portions, where the skin is painful on pressure.

Every part attacked by erysipelas exhibits on the second or third day its highest degree of swelling and redness. In the facial form patients at this time are greatly disfigured and scarcely recognizable; they are unable to open the œdematous eyelids, which are often as tense as a drum, and the external ear-passage and the entrance of the nostrils are also not unfrequently swollen up.

The serous infiltration of the skin is of various degrees. Sometimes it is firmly stretched and has a shining aspect, erysipelas glabrum s. levigatum; sometimes it appears, in consequence no doubt of the manner of its attachment to the underlying parts, warty or wheal-like, e. verrucosum, or, when the streaked form prevails, e. variegatum. The epidermis is often, in consequence of unusually strong distention and rapid exudation, raised up, especially upon the face, in the form of larger or smaller blebs, according to the size of which the older authors distinguished an erysipelas miliare, phlyctænulosum, vesiculosum, bullosum, and pemphigoides.

The formation of these blebs has no specific significance in the disease. Sanson indeed is of the opinion that in the forms described as e. œdematosum and erythematosum, the swelling may, with the aid of a lens, always be found covered with small phlyctenæ. The blebs are filled with a clear, yellowish serous fluid, sometimes tinged with blood, that contains numerous bacteria, and is often gradually made turbid by the abundant appearance of pus-corpuscles, or, according to Lewin, by cells from the rete Malpighii.

On the third, fourth, or fifth day of its existence, the swelling subsides, the redness fades generally to a light-yellow color, and the painfulness diminishes. The tension in any blebs that may be present relaxes, and their contents are reabsorbed, or they burst and dry up into lemon-yellow, semi-transparent scales, or less frequently into crusts of considerable size, e. crustosum. Desquamation of the epidermis, in the form of large or branny scales, and wrinkling of the skin, terminate the local process. There often remain for days or weeks, afterwards a little œdema, a certain stiffness, and a slight anæsthesia of the skin.

Not unfrequently more or less numerous abscesses are formed in the subcutaneous fibrous tissue after the termination of the acute process, which protract the convalescence for months at times, and upon the scalp especially may attain a wider spread. Landouzy counted as many as sixty-nine upon a patient. They do not appear, however, before the complete termination of the dermatitis, which is a circumstance of importance in diagnosis, because in phlegmonous processes the abscesses mainly accompany the course of the disease. They seldom give rise to serious symptoms, but are perfectly reabsorbed, as in Orth's experiments upon animals. Their origin may probably be traced to the little points of suppuration situated, according to Biesiadecki, in the tips of the papillæ.

The hairs fall from the scalp, as a rule, during convalescence, in consequence of the dropsical infiltration, by which the external root-sheath is separated from the vitreous layer of the follicle as far as its junction with the papilla (Haight). The hairs, however, quickly form again.

Generally at the close of the erysipelas the skin returns completely to its normal state. In certain places, however, the skin, on the contrary, undergoes more or less extensive gangrenous destruction, as upon the eyelids (Arlt), the scrotum, prepuce, and vulva; parts, that is, where the particularly thin and tender skin, without any firm underlying tissue, and held tightly at only one edge, is provided with loose cellular tissue, and undergoes, in the course of the disease, enormous swelling; less frequently upon the skin of the cheeks and the patella (Heyfelder), where the circulation has been obstructed by great œdematous distention. A marked bloody hue of the contents of the blebs is often the first sign of commencing gangrene, which may at times be checked by early puncturing. Slight defects are quite rapidly repaired, according to Billroth, but larger ones lead to serious consequences.

The lymph vessels are implicated in erysipelas in rather a specific way. As in every acute, even non-specific disturbance, they swell in the neighborhood of the affected part as far as the nearest glands, and often form cords and knots perceptible to touch or sight. If Béhier and Mittenheimer failed to discover this condition at times, it was perhaps in cases where it was concealed by the thick swelling of the skin. The glandular affection is so much more constant still, that Blandin and others regard the whole process as a capillary lymphadenitis. The slow progress, however, and the limitation of the glandular infiltration to the immediate neighborhood of the affected portion of the skin, are opposed to such a view. Lymphadenitis at a distance from the affected part never occurs, although it accompanies the progress of the erysipelas. The subsequent course is distinguished from diphtheritic and similar affections by the rapid retrogression of the affected glands with the termination of the cutaneous affection. Exceptionally only, three times in forty-two cases as observed by Heyfelder, do chronic infiltrations remain, and suppuration, as in other specific or phlegmonous processes, is very rare. Colin describes a rare case of abscess of the parotid.

These observations substantiate our theory of the slight virulence of the morbid poison, which seems to be inactive after a definite and brief period.

The acute irritation of the glandular apparatus gives rise to leucocytosis (Virchow [1]), which is not very serious when the extension of the process is limited; but in wandering erysipelas, when it affects the whole body in debilitated persons, it may lead to bad results in consequence of excessive irritation of the lymph vessels.

According to the part affected, we distinguish an erysipelas faciei, capitis, auriculare, when it is confined to the ear and its immediate neighborhood; trunci, resp. mammæ, thoracis, abdominis, genitalium, extremitatum, etc. It affects the face and head most frequently, and next to these the lower extremities.

P. H. Bird found that in 260 cases the face and head were attacked 125 times 61 males, 64 females), and the extremities 135 times. In 81 cases of (idiopathic?) cial erysipelas it affected the right side in 56 per cent. of the females and 50 per ent. of the males, the left side in 29 per cent. of the men and 19 per cent. of the omen. It started upon the median line in 13.7 per cent. of the women, and in 1.8 per cent. of the men; upon both sides simultaneously in 5.9 per cent. of the omen and 2.9 per cent. of the men. Of Heyfelder's 42 cases, 28 affected the face, the face and trunk, and 11 the trunk and limbs. In Billroth's surgical clinic at ürich, in the years 1860–70, there were 67 cases of erysipelas of the face, 13 of the alp, 45 of the trunk, 43 of the upper, and 80 of the lower extremities, although rice as many injuries of the lower extremities as of the head were treated. mong Ritzmann's cases 84 were upon the lower extremities, 35 upon the upper, 10 pon the trunk, and 13 upon the head, corresponding to the very small number of juries upon the head and the very great number upon the lower extremities. fleger's observations confirm those of Graves, that erysipelas, when it begins at the iddle line, generally spreads symmetrically upon both sides.

The starting-point of erysipelas is formed, as we have stated, y injuries of the most various kinds, in the skin and the ucous membranes lying near the surface of the body, especilly the points of junction of the latter with the skin.

In Heyfelder's 42 cases it was the nose 12 times, the ear 6 times, eyelid 5 times, pper lip once, lymph glands twice, mamma once, and in the other cases injuries sewhere, of which 5 existed upon the scalp.

The condition of the mucous membrane is of special interest. Vhile it is never attacked primarily in deeply seated parts by erysipelas, the process does often take its origin unquestionably from portions which lie nearer the surface of the body, whether it be from a scrofulous affection, from a diphtheria of the tonsils (Bayer), from a gangrenous ulcer of the larynx (Laborde), from a local inflammation in typhoid fever (Murchison), or from a traumatic lesion, as the tearing out of nasal polypi (Volkmann), the wounding of the vagina during delivery (Hervieux), and the like. Trousseau was even of the opinion that facial erysipelas generally started from an angina.

---

[1] Cellularpathologie 1871. p. 230.

The extension of the process to the outer skin takes place by continuity of tissue, either directly from the mucous membrane of the mouth, or through the posterior part of the nasal cavity and the tear-passage, through the lower nasal entrance, or through the Eustachian tube and the external auditory passage.

The process may also remain wholly confined to the mucous membrane; at least this may be inferred from the eighteen cases of angina collected by Cornil, which occurred at the time of an epidemic of erysipelas at the Paris hospitals. Cornil designates, as the characteristic of such an erysipelatous angina, the purple-red, shining, œdematous swelling of the whole pharyngeal mucous membrane, which looks as if varnished, while the tonsils may remain free. Great pain is produced by swallowing, and when the inflammation extends farther forwards salivation ensues. The glands of the lower jaw and neck are much swollen. Occasionally blebs are formed, which quickly dry upon the mucous membrane, and leave behind a softened, corroded epithelial layer; in some cases it runs into gangrene.

The diagnosis from other similar affections designated by Lewin as pharyngitis sicca is seldom possible; it can only be determined with certainty when the erysipelas spreads to the external skin. It is probable that many of the observations concerning fever, which precedes erysipelas one or two days, relate to cases where the affection has been limited for a time to the mucous membranes.

In a patient of Brochu's [1] there existed for five days a severe pharyngitis, with swelling of the lymph glands. He was discharged, but returned to the hospital after a few days with facial erysipelas. It was only possible by this second observation to determine the nature of the affection of the mucous membrane.

---

[1] Gaz. d. hôp. 1874. 13 Jan.

Sometimes the disease spreads almost simultaneously over the mucous membrane and the skin.

Jules Simon saw an erysipelas of the face and scalp starting from an angina, in which the inflammation was so severe that the jaws were closed as in trismus. The patient could not swallow, was at first hoarse, finally voiceless, and died delirious on the tenth day, the skin of the face and neck being of a bronze color. On dissection the posterior parts of the mucous membrane of the mouth were violet red, and that of the pharynx thickened and softened; in the air-passages, the mucous membrane of the epiglottis, larynx, vocal cords, of the trachea and the bronchi as far as their finest divisions, appeared as red as if soaked in blood, but free from secretion.

The mucous membranes bordering upon the skin are also secondarily attacked by an extension of the erysipelatous inflammation. The mucous membrane of the nasal and pharyngeal avities is found, in erysipelas of the head and face for instance, generally hyperæmic and swollen; sometimes the palate and auces, and less often the tonsils, are so swollen that swallowing s difficult (Arnott, Wutzer, Trousseau, Oppolzer). Among ixty-five cases in Wunderlich's clinic, Blass saw pharyngitis hirty-two times, and in nine cases so severe that without other erious general symptoms difficulty of swallowing ensued.

Semeleder[1] found an inflammatory redness and swelling of he epiglottis and entrance of the trachea, without subjective rouble and change of voice, which gradually disappeared with he progressing desquamation of the skin, but returned with a elapse.

Not very unfrequently the affection reaches a higher degree and extends farther. In several instances by Velpeau, Bayer, Gubler, Dujardin-Beaumetz, and others, an œdema of the glottis was rapidly developed, that often rendered tracheotomy necesary, which, however, was seldom successful. Lewin found, on dissection of such a case, swelling of the arytæno-epiglottic folds and of the posterior wall of the larynx, with intense redness of the vocal cords. Such processes may become, according to his views, the starting-point of new growths upon the vocal cords.

---

[1] Die Laryngoscopie und ihre Verwerthung für die ärztliche Praxis. Wien, 1863.

The erysipelatous inflammation of the mucous membrane, whether of primary or secondary origin, may also assume a wandering character, and from a pharyngitis extend to the air-passages farther down, and even produce pneumonia. Former writers regarded this as erysipelas internum. Without referring to these, however, Peter, Trousseau, Labbé, Jules Simon, and others mention cases where pneumonia has arisen from an erysipelatous angina through descending laryngitis and bronchitis of the large, smaller, small, and smallest bronchi.

Labbé describes a case of facial erysipelas which appeared four days after the beginning of the fever. During this time the patient had a considerable angina, hoarseness, and swelling of the mucous membrane of the mouth, so that an erysipelas starting from the pharynx was recognized. The patient died on the eighth day. Extensive pharyngitis was found; the inflammation extended downwards over the epiglottis and larynx; the trachea and bronchi were deeply reddened; pneumonic infiltrations existed at the base of both lungs, and the bronchial branches leading thither were acutely inflamed.

This possibility of the direct extension of the irritation by continuity of tissue to the mucous membrane of parts even below the vocal cords is peculiar to erysipelas. In affections apparently similar, diphtheria and the like, the vocal cords more frequently form the natural limit to the spread of the inflammation.

Organs of Digestion.—Somewhat severe disturbances of the digestive apparatus show themselves as a nearly constant symptom of erysipelas. The tongue is covered with a white creamy coat, which dries gradually, becomes of a dirty yellow color, and, when the fever is protracted, blackish and crust-like. The taste is pap-like, the appetite fails, and the thirst is much increased. Great nausea, and even repeated vomiting, often accompany severe cases, especially of erysipelas of the face and head. These symptoms led the older physicians to the theory of a bilious erysipelas; and Béhier still refers cases where bilious vomiting and a yellowish hue of the face are present to this class. The urine is also said to be colored by bile at times. General jaundice, however, is seldom observed, and Lebert suggests that the nausea and vomiting may possibly proceed from the giddiness of the head in severe erysipelas.

The liver seems to be generally somewhat enlarged, and is more or less sensitive on pressure. The spleen also is enlarged, but the swelling disappears, as a rule, with the termination of the cutaneous inflammation (Friedreich).

Intestinal Canal.—The discharges from the bowels are not materially altered, but are occasionally diarrhœal in character; the cases of intestinal hemorrhage and of ulcers in the duodenum, however, which rarely occur, and which prove fatal from profuse hemorrhage, are very remarkable. The various instances have occurred in cases of facial erysipelas, which were in no way of great severity. They suggest the similar symptoms which are occasionally observed after burns of the skin, and bear the same explanation.

A case of this sort, described by Malherbe, was that of a man twenty-four years ld, in whom the disease ran its course with severe nausea and vomiting. On dis-ection, the stomach and the duodenum were found hyperæmic to a marked degree, he intestinal mucous membrane elsewhere considerably swollen, and on Kerkring's olds, fifty to sixty ctm. from the pylorus, were several ulcers, some of them as auch as a ctm. in diameter, the borders of which were not swollen. In two other ases of Larcher's, in men fifty-two and sixty-eight years old, the disease lasted eleven lays; there was severe bilious vomiting, and in one case constipation and subse-uently diarrhœa. On dissection several ulcers (four) were found around the open-ng of the ductus choledochus and pancreaticus, and in one case, even a fifth, on the osterior intestinal wall, close by the pylorus, all of quite regular, oval form, and onfined to the mucous membrane simply. In the neighborhood were numerous cchymoses. The intestinal glands were not swollen. Bayer also observed a case f erysipelas starting from the upper part of the nose after diphtheria of the tonsils, rhich extended over the forehead, and on the fifth day of its existence, during a rofuse intestinal hemorrhage of which the patient died, disappeared. There was ound on dissection a peritoneal transudation colored with blood, the peritoneal coat f the ileum tinged with blood, the contents of the lower part of the small intestine s far as the valve markedly bloody, and an abundance of tar-like matter in the arge intestine. Bayer is inclined to refer these appearances to a transitory conges-ion of the intestinal mucous membrane. Schönlein states that in times of extensive pidemics many otherwise healthy persons living within the affected district were ttacked by severe diarrhœa, without the occurrence of any erysipelatous process pon the skin.

Kidneys.—The urine is generally scanty and turbid at the height of the disease, and contains albumen, which, according to Lebert, may persist for weeks even after recovery. In severe cases casts and hemorrhagic elements are found. Lebert is of the opinion that possibly a permanent nephritis is produced by the erysipelas, and Desprès would even refer uræmic symptoms to a preceding erysipelas, an opinion which is not supported by evidence in our possession.

Cerebral Symptoms.—Patients complain most of giddiness and of more or less severe headache, which is especially violent in erysipelas of the head in consequence of the great stretching of the skin. They are excited, anxious, and very sensitive to external impressions upon the organs of hearing and vision. Sleep is uneasy, often disturbed by wild dreams, and sometimes wholly absent. Mild delirium accompanies nearly all cases in which a large extent of skin is involved, and in erysipelas of the head and face it is not unfrequently furious, the patients shrieking, springing out of bed, and the like.

The epidemic described by Schönlein was characterized by severe cerebral symptoms of this sort, which generally lasted several days. In a patient of Volkmann's they were so violent that the use of the strait-jacket was required. A case is mentioned by Colin, of a man twenty-five years old, in which the delirium lasted ten days, with coma, subsultus tendinum, and involuntary dejections; the patient, however, recovered. Holm reports a similar case, which relapsed after an interval of three weeks, and, like deep-seated affections of the central nervous system, terminated in a symmetrical gangrene of the distal phalanges of the second, third, and fourth fingers, with idio-muscular contraction on pressure of the pectoralis major, deltoídeus, and biceps.

In a *prognostic* sense this condition, which is probably produced mainly by the cutaneous irritation, has no bad significance. It disappears, as a rule, simultaneously with the abatement of the cutaneous symptoms. In those who have died during severe cerebral symptoms, Trousseau and others have been unable to discover any considerable changes in the brain and its membranes. It is possible that the impediment to the venous circulation, produced by the swelling and infiltration of the parts, may assist in the production of the delirium in erysipelas of the head and face.

Fever.—In the majority of cases (in all of Heyfelder's and in two-thirds of those of Ritzmann) a more or less severe chill (less frequently several milder ones, occurring one after the other) marks the beginning of the disease. In other cases (twenty-seven per cent. according to Ritzmann) the initial chill is wanting, especially, according to Volkmann, in patients who have previously exhibited considerable febrile symptoms, and where also the sudden rise of temperature produced by the erysipelas is very inconsiderable.

Generally, as in recurrent typhus, the temperature very rapidly, often within eight to twelve hours, reaches the point of 40° C. [= 104° F.], or higher. A more gradual elevation takes place less frequently, any considerable height not being reached before the second or third day (Wunderlich).

Corresponding to the remission described by Thierfelder in typhoid fever, the temperature exhibits at times, after the appearance of the exanthem, a marked depression, which may last a certain time, a day even, and then quickly rise again. Generally, however, the temperature remains at its height, and even increases during the following days, so that the exacerbations soon mount between 40° C. [= 104° F.] and 41° C. [= 105.8° F.], and in some cases even 42° C. [= 107.6° F.], according to Wunderlich and others.

The subsequent course of the fever depends exclusively, in uncomplicated cases, upon the extension of the exanthem. When the process spreads quickly, uniformly, and to an intense degree upon the skin, the fever becomes continuous, with very slight remissions, which often amount scarcely to 0.5° C. [= 0.9° F.]. The time of the remissions varies considerably ; they generally occur in the morning, or, when the evening exacerbation is followed by a secondary one in the night, in the later forenoon hours. In other cases, especially in those in which the spread of the erysipelas takes place more slowly, there occur, after high evening temperatures with frequent secondary exacerbations, marked diminutions, not unfrequently accompanied by perspiration.

Finally, in those forms where the erysipelas progresses by extensive and irregular advances, and is protracted for weeks, the course of the fever is apparently irregular, although probably parallel with that of the cutaneous affection. Corresponding to these advances, which are broken by long and irregular intervals, it often appears to be interrupted by very considerable remissions, and even complete intermissions, which are followed by more or less rapid and prolonged elevation of temperature.

The decrease is generally as rapid as the ascent; and in many cases is preceded by a perturbatio critica (Thomas). The temperature sinks continuously in a few hours, or in a single night, to, or nearly to, its normal standard. Fluctuations even then follow at times. Less frequently the fall assumes a more remitting character, the diminution of temperature being distributed more or less uniformly over several days.

The duration of the fever, exclusive of complications, is determined solely by that of the exanthem. Neither the high temperatures nor the duration of the fever are of evil significance as regards prognosis, except in patients previously debilitated; even very high grades of temperature are relatively well borne. Death takes place generally during high fever (Wunderlich); seldom at the temperature of collapse, and is often, according to Wunderlich and Eulenburg, followed by a prolonged post-mortem elevation of 1.2° C. [= 2.16° F.] to 1.8° C. [= 3.24° F.].

Cases occur also of very slight fever, accompanying an exanthem of mild intensity, which may be regarded as abortive forms, especially in times when erysipelas prevails.

The *pulse* generally corresponds in frequency with the elevation of temperature, 100 to 120, and not unfrequently 140 in the minute. In quality it exhibits nothing especially characteristic, varying according to the greater or less strength of the patient. Many authors (Lebert and others) describe it as especially soft; and when the erysipelas has existed for a long time, and there are slight remissions, it is often considerably smaller and weaker; and in many cases also intermittent and dichrotic. A marked increase in frequency after the fifth or sixth day is, according to Nunnely and Hinckes-Bird, of unfavorable significance. After the diminution of the fever, its frequency generally falls for a time below the normal rate.

The action of the heart corresponds with the pulse. Great akness of both is an important indication in treatment. In ost cases the first sound is accompanied by a systolic murmur, ard most distinctly at the apex, which, according to Jaccoud, independent of the high degree of fever, and disappears ter its termination. Sevestre observes that endocardial prosses sometimes occur in the course of severe cases, which perst after the disease is over ; his theory, however, is based solely n the systolic murmur. One of his cases terminated in fibrinus pericarditis ; and Durozier describes a similar case.

Complications and Sequelæ.

Hypostatic *pneumonia* sometimes occurs, especially in erypelas of long duration (Volkmann). Some cases of severe oupous pneumonia belong to a post-febrile stage (Trousseau, itzmann).

Pleuritic affections seem to be more common, and it is a uestion whether they are to be regarded, like other phenomena, s the direct continuation of the process to deeper parts. Lawnce, Wutzer, and lately Volkmann, consider them as such, ecause they appear especially with erysipelas of the chest, and ecause a suppurative or hemorrhagic inflammation in the nterior mediastinum was also observed under the same cirumstances in certain cases (Lawrence, Ponfick). On the other and, Daudé mentions a pleurisy which complicated facial erypelas, where, as in a case of Ritzmann's, no continuity of the rocess could be shown.

Several cases of *peritonitis* have also been described. Whether hey are to be regarded as an immediate extension of the inflammation from the abdominal walls, cannot be decided from the material thus far collected. Samuel Wilkes,[1] among others, efers a case under his observation to this cause, and classes ith it another case, in which acute general peritonitis appeared fter a burn upon the abdomen. Ritzmann recently observed a eritonitis complicated with pleurisy and pyothorax in erypelas of the trunk, without being able to decide in favor of his theory.

---

[1] Guy's Hosp. Rep., 1861.

Older writers, Martinet, Parent-Duchâtelet, Lietzau, and others, often regarded a *meningitis* as an accompanying symptom of erysipelas cap., which is supposed to arise readily in consequence of violent suppression of the cutaneous inflammation by the use of cold and moisture for instance, that is, by metastasis or repercussion. Trousseau, too, looked upon it as a not infrequent complication of "epidemic" erysipelas, although not occurring in the "sporadic" form. The theory rests, however, wholly upon the violent cerebral symptoms during life; it has not been established by dissection. Trousseau failed to find it in the examination of persons who died with cerebral symptoms. Exclusive of severe cranial injuries, I find recently only the case of Rhode, who describes a tubercular meningitis ending fatally five months after the termination of a facial erysipelas. It was apparently connected with caseous degeneration of hemorrhagic infarction of the lung, the relation of which to erysipelas, however, was not to be made out. A primary suppuration in the immediate neighborhood of the cranial bones, as, in an instance of Lebert's, within the frontal sinus, may serve as the starting-point simultaneously of meningitis and erysipelas.

Pyæmia is extremely rare in uncomplicated cases, according to the data before me. The evidence which has been collected in support of the widely spread opinion to the contrary, appears to me to have been derived from cases in which the erysipelas was associated with severe or extensive injuries. The pyæmia here is apparently of great assistance, because, in accordance with the view of Volkmann, it appears to favor the destruction of any thrombi that may be already present, and the destruction of embolous infarcts. The great debility after prolonged erysipelas may in such cases also easily give rise to venous thrombi.

On this theory the rare cases of multiple inflammations of the joints, which occur at a distance from the seat of the erysipelas, and of which earlier writers (Lawrence, Velpeau, Avery) furnish some examples, may be referred to pyæmia.

Desprès (Traité de l'Erys. Paris, 1862) saw a case, in a facial erysipelas that arose from an operation for cataract; others, seen by Volkmann and Ritzmann, affected only the severely wounded.

Besides these cases, Ritzmann observed patients in whom an sipelas of the extremities in the neighborhood of joints super-ally situated (the ankle, knee, and the like) led to inflamma-n of the same. Rayer had also made similar observations, d a recent case from Volkmann's clinic is described by Anger-ausen. This is not, however, as some believe, an extension of he inflammatory process from the skin to deeper parts, and o the joint. Ritzmann draws attention to the fact rather that the affection occurs only as a complication of severe fractures, especially those produced by gunshot, and attacks not the wounded joint, but generally that situated nearest the injured part. A great œdematous swelling as the result of erysipelas may, in consequence of the strong disposition of such injuries to secondary inflammation of the joints, easily give rise to such, as may other relatively slight irritations.

On the other hand, extensive gangrene of the skin in consequence of erysipelas not infrequently points to severe pyæmic and septicæmic phenomena, generally of evil omen, of which literature furnishes many examples.

Malmstén (Hyg. 21) describes a thrombosis of the portal vein after an erysipelas gangrænosum, which extended over the abdomen, sides, and back as far as the axillæ upwards, and downwards over the thigh and scrotum, and caused gangrene of the latter. The patient, twenty-four years old, died on the sixth day of the disease, with typhoidal symptoms, coma, delirium, and the like. On cutting through the abdominal integuments, where the erysipelas was seated, the subcutaneous cellular tissue was found swollen, loosened, and pervaded with pus, which had nowhere collected in masses. The skin of the erysipelatous half of the scrotum was gangrenous, with a superficial line of demarcation. A great quantity of ichorous matter flowed from the underlying cellular tissue on cutting into it. In the abdominal cavity were several large spoonfuls of clear serum. The peritonæum was actively injected only upon the last coils of the ileum and cæcum, corresponding to the intestinal mucous membrane at the end of the ileum and cæcum. The spleen was enlarged, spongy, and soft. The liver was greatly enlarged, anæmic, and fatty. The vena porta was completely distended by a particularly firm, fibrinous coagulum, which extended into its ramifications within the liver. This lay so loosely in the vessels, that it could be easily drawn out. The coats and the inner surface of the vessel were normal. Incipient fatty degeneration of the kidneys was found.

In a case lately observed by Tutschek, a facial erysipelas led to a peculiar stopping up of the abdominal aorta at its bifurcation in consequence of true thrombosis of the heart. The rarity of the affection makes the communication of the case pardonable, even if its explanation is wanting.

H. W., a soldier, æt. twenty-two, was received into the hospital on the 12th of February, 1873, with an erysipelas of the right half of the face, which had started the day before from a pustule upon the right ear. There was high fever. Painting the parts with collodion was ordered. The erysipelas extended over the left half of the face, the back of the head, and neck, diminishing in intensity. On the evening of the fifth day the temperature was normal, and the pulse, previously full and hard, was eighty-four, small and weak. A slight systolic blowing was heard at the apex of the heart. There was delirium. On the succeeding days the pulse was stronger, the delirium abated, and the appetite returned. On the morning of the tenth day of the disease, the patient having complained since ten o'clock on the preceding evening of a sudden attack of violent burning, tearing and stretching pains, with coldness of the legs, and difficulty in moving them, the feet, the legs, and the lower third of the thighs were found to be stone cold. The skin of the same was covered with purplish spots, and its sensibility was impaired; the feet were entirely deprived of the latter and of the power of motion, and passive movements caused intense pain. There was no pulsation in the femoral artery. The radial pulse was quite strong. The action of the heart was increased in force, 100, and the first sound was distinct and accompanied by a blowing murmur. The patient died twenty-six and three-quarters hours after the beginning of the first symptom of arterial stagnation. Dissection.—In the left heart, in the depressions between the columnæ carneæ, toward the apex and behind their point of insertion, were numerous closely attached, firm, yellowish-white, fibrinous coagula, varying in size from a pea to a filbert, with fringes which floated free under water; some of them were less firmly attached, and could be easily torn off with forceps. The endocardium was normal, granular degeneration of the transverse fibrillæ being recognized only in places. On the free borders of the semilunar valves there were small, detachable coagula of fibrine. The inner surface of the aorta was smooth and soft. In the left renal artery there was a small thrombus running into both branches of the vessel, which entirely filled up the lower one as far as the renal substance, but the upper one only imperfectly. The lower half of the kidney was correspondingly enlarged, colored yellow, and in a state of fatty degeneration. A thrombus completely filling the vessels was found in the abdominal aorta and its branches, beginning five ctm. above the bifurcation, extending nine ctm. into the common iliac arteries as far as Poupart's ligament, and several ctm. into the hypogastric arteries. None were found in the other branches of the aorta. In the spleen, which was twice the natural size, there was a wedge-shaped, fibrinous infarction as large as a walnut.

The *eyes*, in facial erysipelas, readily suffer by sympathy, Conjunctivitis and photophobia accompany the majority of cases, but quickly disappear after the termination of the affection. Ulcers upon the cornea sometimes occur (Anstie), the softening and perforation of which may lead to atrophy of the bulb (Wagner). Cases in which the erysipelas proceeds from an abscess behind the globe are the most serious. In a patient of Arlt's, iritis occurred, with great exudation, followed by shrinking of the globe after the former was evacuated. Glaucoma (?), neuro-retinitis, and atrophy of the optic nerve and of the retina are also described. Certain disturbances of vision, which occasionally follow erysipelas, are produced, however, according to Mathis, solely by movable opacities of the vitreous. Gangrene of the eyelids, according to the communications of Arlt and Biermann, is apt to lead to complete suppuration of the globe.

According to some observations by H. Weber and Mugnier, *mental disturbances* may remain after the termination of erysipelas, especially maniacal delirium, with delusions or melancholy, which offer a favorable prognosis and quickly disappear.

A peculiar effect is often exercised by erysipelas upon preexisting chronic or acute morbid processes in the *skin*. Cazenave, Schedel, Sabatier, and others have observed, that after the termination of a casual attack of erysipelas, old eczemas, lupous affections, and ulcers upon the legs are quickly healed. In several cases reported by Desprès, Champouillon, Mauriac and others, extensive phagedænic and serpiginous chancres, and even chronic abscesses produced by caries of the bones, which had previously defied all treatment, healed rapidly during an erysipelas. Even extensive non-malignant tumors may be made to undergo absorption, according to the observations of Legraud, Busch, and Volkmann, by erysipelas, although the cure does not seem to be always permanent.

Desprès was induced by these results to recommend the artificial production of erysipelas as a means of cure for phagedænic ulcers (!).

On recent wounds also erysipelas often seems to exert an influence, although not always one favorable to their recovery; especially, according to Ritzmann, where small wounds still exist. In five of his cases, deeply penetrating wounds, they were attacked by gangrene, which they recovered from, however, after the termination of the erysipelas.

The explanation of this peculiar influence, which is observed also in other violent cutaneous inflammations, in variola for instance, is possible on the supposition that old infiltrations may be more easily brought to softening and resorption by the uniform, intense inflammation of the skin and the altered relations of tension and hyperæmia in the affected parts.

Diagnosis.

The diagnosis of erysipelas is generally certain as soon as the eruption has shown itself upon the skin. The rose-red color of the painful, hot, œdematous, shining, and swollen portion of skin, generally with a sharply defined border upon one side, and starting from the opening of some natural cavity, or from a wound; the similar inflammation of neighboring mucous membranes; the swelling of the lymph glands, the rapid and marked elevation of temperature, the serious general symptoms, and the favorable ending in desquamation, are sufficiently characteristic.

On the other hand, erysipelas occurring primarily upon the mucous membranes, especially of the pharynx and nasal cavities, escapes recognition until it has extended to the external skin. Swelling of the glands, and serious constitutional symptoms at the time of epidemics, should direct attention to this.

Simple *erythema* (E. simplex), which is a transitory hyperæmia of the cutis, runs its course without marked increase of temperature, without pain and swelling of the skin and glands, and leaves no desquamation or pigment deposit behind. Erythema nodosum is characterized by exudation in the deeper parts of the skin, particularly in the neighborhood of the bones, which, however, is always sharply defined in the form of papules, nodules, or nodes.

Urticaria is distinguished from erysipelas by its peculiar nettle-like itching, the wheal-shaped eruption, and its simultaneous extension over more or less extensive portions of the body.

The acute superficial *lymphangitis*, which is accompanied by swelling, redness, pain, and fever, has a streaked or spotted, seldom a confluent, redness; its extension is always centripetal, the stretching of the skin is moderate, and the inflamed lymph-vessels appear like firm cords. Its termination in resolution is less frequent than in suppuration.

The course of *diffuse phlegmonous inflammation*, often described as pseudo-erysipelas, or erysipelas phlegmonosum, exhibits at first high fever, painfulness, redness, and swelling of the skin and of the glands. The redness is here much darker, and nowhere sharply defined, and the cutaneous swelling is of board-like hardness, and generally ends in suppuration, in which case the abscesses in the subcutaneous cellular tissue quickly become confluent.

The differential diagnosis of erysipelas from a *progressive and diffuse purulent inflammation of the cellular tissue*, and from an *acute suppurative œdema*, which spreads rapidly and extensively from severe injuries, with irregular, diffused, and often dirty redness and œdema, is not always easy. Of decisive importance in this respect are the slow increase of the fever, the rapid development of purple or discolored vascular networks, the doughy quality of the swelling, and the peculiar ichorous or gangrenous character of the wounds, while the general condition of the patients may remain for a long time without serious subjective disturbances (Volkmann).

The *œdematous form of charbon* (œdema malignum s. carbunc.) occurs only in places where the skin is very thin, on the eyelids, on the neck, and in the axillæ, seldom on the extremities. The swelling comes on quickly, and is extensive, so that the lids, for instance, are often in contact with each other by their external surfaces, like two shining, semitransparent pads. The swelling, however, soon becomes hard and uneven, and blebs filled with sanguinolent fluid are developed upon the swellings. The painfulness is limited to the swollen lymph-vessels, and severe fever occurs only when the disease has existed for a longer time. The affection ends with sloughing, or fatally with typhoidal symptoms.

Pemphigus is distinguished from erysipelas bullosum by its more or less uniform localization and the fixed character of the cutaneous inflammation, which progresses without œdematous swelling, and without constant participation of the lymph-vessels and glands.

With regard to the extensive epidemics of *ignis St. Anton.* (called after the foundations of this order), which prevailed in central Europe in the tenth and twelfth centuries, and which, as Daudé states, are described, according to the theory of Cullen and others, as erysipelas gangrænosum or putridum, no definite judgment can be given.

Stages and Duration.

A division of the erysipelatous process into distinct periods is not practicable. Every affected portion of the skin is subjected to a typical course, in which the stages of incubation, efflorescence, and desquamation may be distinguished. Inasmuch, however, as the affection does not remain limited to the place first affected, but advances more or less irregularly, all stages of the dermatitis are generally found simultaneously near one another, and accordingly the general symptoms are often destitute of determinable divisions. On this account the duration of the disease is also very variable. In erysipelas of the face and head it amounts, on an average, to ten, twelve, or fourteen days, less frequently to six, but sometimes to more, even two, three, and four weeks, and even two or three months when it extends over large portions of the skin.

Most of the statements of writers upon the various epidemics lie within these limits. As the average number of days, Velpeau gives twelve, Heyfelder thirteen and a half, while Billroth considers fourteen days as exceptional even. The longest duration is shown when the trunk is attacked primarily or secondarily, the shortest, in erysipelas of the lower extremities, in which, according to Volkmann, the fever may disappear after thirty-six hours even. Ritzmann found the average to be five days when the erysipelas was limited to a portion of the head, 7.6 when the whole head was affected, and in cases starting upon the head 7.3; in the forms which extended from the trunk 9.5, in those starting from the upper extremities 8.2, and in those from the lower extremities 8.4 days. There are but few observations concerning the duration of the period of incubation.

Convalescence is generally short and quick, but it may be greatly prolonged by complications

Relapses.

Persons who have once had the disease retain for it, in con:ast with other infectious diseases, a very great susceptibility.

In many surgical cases a patient undergoing protracted suppuration may be ttacked with erysipelas two or three times during the process. Volkmann reports everal instances of this. Of Ritzmann's 146 cases of erysipelas in the Berlin Miliary Hospital, 13 per cent. relapsed; of 248 cases in the Zürich surgical clinic 7.6 er cent. relapsed, and there were two cases in forty-two of Heyfelder's. Of the ormer, fifteen were attacked twice, and two three times with erysipelas during heir residence in the hospital. Heyfelder reports the case of a patient with angrene, in whom an erysipelas relapsed seven times in seven months, extending ach time over the whole body as far as the fingers and toes. He became emaciated o a skeleton, but finally recovered.

The later attacks generally run the same course as the first, ut sometimes the constitutional as well as the cutaneous sympoms are milder. Repeated relapses on the same part may lead o permanent thickening of the skin.

Not a few persons are subject to a periodically returning rm—the so-called *habitual erysipelas*, which affects mostly ie face or lower extremities. The relapses occur once or twice year, and appear to start from chronic ulcers of the nasal ucous membrane, of the tear-passage, or of the leg. If scroulous persons are more frequently attacked than others by it, is the more frequent occurrence of ulcers of the mucous memrane among them that is the cause. The course of the relapse, ie contagiousness of which in certain cases has been established ee above), does not vary materially from the primary process. s spread is generally confined, however, within narrow limits; ecause, as I believe, the integument, which is thickened at its orders in consequence of the repeated inflammations, offers an bstacle to the farther advance of the transudation. Occasionlly, too, they appear to give rise to pachyderma. Schwalbe, of ostariea, describes an "hereditary" erysipelas occurring in iree generations of a family, which led to elephantiasis.

The expression "habitual erysipelas" has been often used very loosely in erature. Many of the forms described under this name have certainly nothing in common with true erysipelas, except some external appearances. With many women, for instance, erysipelas is supposed to occur regularly in the place of the catamenia, or simultaneously with them. With regard to some of the older descriptions, indeed, a safe opinion cannot be expressed; but many of them relate, as Volkmann shows, merely to acute œdematous affections of the skin, without, or with but little, redness, and without a disposition to wander. Kaposi also makes a definite distinction between true habitual erysipelas which starts in lupus vulgaris from the cauterized points, and in lupus erythematosus from the places on the face and ears where the acute eruption is especially localized, on the one hand, and on the other hand a streaked or striped lymphangitis and a peculiar superficial infiltration of the cutis, often accompanied by typhous symptoms, in lupus erythematosus. He invests the latter with the inappropriate name erysipelas perstans faciei, although it is quite different from the specific erysipelatous inflammation of the skin.

Varieties.

Erysipelas neonatorum, which most commonly occurs upon the navel and other parts of the abdomen, was formerly regarded as a special variety. It presents, however, no special and specific exceptional peculiarities. Its cause is to be referred, according to Billard, partly to the easy vulnerability of the hyperæmic skin in the first days of life, after the removal of the vernix caseosa, and partly to the navel remaining open or suppurating, its normal cicatrization being, according to the observations of A. Vogel, especially disturbed during epidemics of puerperal fever. In children somewhat older, erysipelas often proceeds from vaccine or impetigo pustules, from simple wounds, intertrigo, mastitis, ulcerations on the genitals, and the like. It occurs most frequently between the third and tenth day after birth, and afterwards in gradually diminishing frequency. In some foundling and lying-in hospitals it is especially common, but less frequent under the more favorable conditions of private practice.

In proportion to the deficient development of definite bands of fibrous tissue in the skin, the attachment of which to the substratum is still very loose, erysipelas in small children shows a tendency to spread rapidly over the whole trunk, the extremities, and the face. It results, also, oftener than in older persons, in gangrene or prolonged suppurations, the prognosis of which is very unfavorable. Bouchut, Moreau, and Trousseau regard ie affection as always fatal. Steiner (Compend. 1872) had only wo recoveries among sixty cases; Billard, among thirty cases, epresenting the whole early period of life, had sixteen that were atal; while Bednar makes the prognosis better. It appears to e more unfavorable the nearer to its birth the child is, and ecomes better with increasing age. The affection often runs a atal course, with severe febrile symptoms, in a few days; or, fter the erysipelas has wandered for weeks over the skin, death occurs from exhaustion; or, with symptoms of icterus neonat., in consequence of umbilical phlebitis. On dissection, especially in erysipelas of the abdomen, peritonitis is found; and in other ases abscesses in the lungs, liver, spleen, and between the musles, suppuration of the umbilical vessels (Osiander, Martin) and he like are discovered.

There is sometimes described as a peculiar variety, moreover, *rysipelas vaccinale*, that starts from the cutaneous lesions connected with vaccination, but presents otherwise no peculiarities. Vith young children it may easily become dangerous, but ith older persons it is not of serious importance. During n epidemic of erysipelas in Boston, in 1850 (reported in the *merican Journal*, in October of that year), it occurred so freuently in vaccinations that they had to be given up. Eulenerg also reports two cases of vaccinal erysipelas in sailors, hich proved fatal, and advises, therefore, at times when the equent occurrence of erysipelas among the people is observed, at vaccination should be postponed when practicable.

Erysipelas puerperale, too, which was formerly described as n affection especially to be dreaded, has nothing specific. any cases described under this name, like those of Osiander nd Ratzius, correspond to the erysipelas malignum puerperale it. described by Virchow[1] as the result of ichorrhæmia, which e himself considers as a phlegmonous inflammation, which xtends after the manner of erysipelas. True erysipelas is not ery common in childbed, and in lying-in women otherwise ealthy it is without serious import. Its starting-points, accordng to numerous observations (Hervieux, Doublet, Cornil, and others) seem to be here, as in idiopathic erysipelas, various injuries, rents in the vagina, bruises of the vulva, eczema of the nose, an angina, and so forth.

---

[1] Virchow's Arch. XXIII.

Mortality and Prognosis.

The mortality in persons previously healthy is generally light. It is difficult, however, to obtain proper data on which to determine this point, as so few cases are "pure;" the majority of those reported being complicated by other and often severe affections.

Leaving out of the question the element of surgical casuistics, it is not easy to decide in single cases whether the primary processes or the erysipelas were the cause of death. Nevertheless it may be allowable to give certain data, which will at least afford a general idea of the dangerousness of the disease. In nearly 10,000 cases of various authors, surgical included, the mortality was on an average eleven per cent., but varied considerably in the experience of the various observers. Wunderlich had only three per cent. fatal cases (Blass), Volkmann five per cent, P. Hincks Bird seven and a half per cent., Ritzmann in Berlin seven and four-fifths per cent., Williams and Heyfelder in Petersburg ten per cent. In the American war, the mortality of the affected (in surgical cases) was eleven per cent.; whilst individual observers, as Pujos, give thirty per cent. of fatal cases, and Desprès even over fifty per cent.

The diversity in the character of the various epidemics is, in part at least, the cause of these differences, while it may be referred also to the physical condition of those attacked. Like every febrile complication, erysipelas is to be regarded as a serious matter in pre-existing severe general affections. From its occurrence in exanthematic typhus for example, Murchison lost thirty out of ninety-two cases, and in typhoid fever it caused death in six of Louis's nine cases, and four times in the same number of Murchison's. It is also dangerous in a high degree in severe injuries, prolonged suppuration, carcinoma, phthisis, dropsy after nephritis, and the like, especially in the very young or the very old. In debility of the heart's action, also, extensive erysipelas may easily lead to fatal collapse.

Whether *sex* has any special influence over the mortality, cannot be determined, owing to insufficient data. Very young children and old persons appear to succumb to the disease more readily than strong persons of middle age. Many external conditions, too, as the unwholesomeness of certain hospitals, probably have an important influence upon the result.

The *prognosis*, then, in uncomplicated erysipelas, especially in sporadic cases, which are not associated with other severer affections or injuries, is favorable. Fever, even accompanied by high temperature and of long duration, is not of itself a bad sign, except it affect cachectic persons or those with marasmus. Much more dangerous, on the other hand, is erysipelas when it spreads widely over mucous membranes, especially those of the organs of respiration.

In hospitals the mortality may be increased by the great number of cases. Traumatic erysipelas especially gives rise to pyæmic infection in injuries and in childbed. Young children and old people yield more readily to the disease, while stronger persons of mature age resist it better.

The *fatal termination* may be produced in debilitated and otherwise seriously diseased persons by inanition, and perhaps also by collapse; also in consequence of pleuritic and peritonitic affections, by pneumonia, laryngitis, and œdema of the glottis. In severe injuries, pyæmia as well as gangrene of the skin may assist in causing death.

TREATMENT.

Etiological investigations point to the necessity of adopting certain prophylactic measures, notwithstanding the relatively slight danger attendant upon erysipelas, as soon as the disease breaks out or individual cases are introduced into hospitals and similar enclosed institutions. A pedantic cleanliness, such as Billroth observes, immediate and thorough removal of refuse, and especially of soiled bandages, are necessary on other grounds. Gosselin and others advise the isolation of patients as far as possible; and this measure, which is desirable in medical wards, is indispensable in surgical and lying-in establishments. My colleague, Herr Oberstabsarzt Starcke, informs me that erysipelas in the surgical wards of Charité is now confined to the cases brought there as such, since they have followed the practice of isolating them at once.

In a curative sense the treatment of erysipelas has undergone great changes. For a long time is was held that, in addition to the local, a specific general treatment was necessary, on the theory that the process was really a general exanthematic affection.

Venesection figured conspicuously. "Ubi primum accedo," says Sydenham, "satis largam sanguinis quantitatem e brachio extrahi præcipio." The apparently favorable results contributed to keep this means in use for a long time. Copland, Andral, and others were the first to advise against it, because nervous and cerebral symptoms were easily developed in consequence of it, especially in weak persons. ("In erysipelas," says Copland, "there is always a great tendency to asthenic vascular action and to exhaustion of the vital powers.")

After this the revulsive method, the administration of emetics and purgatives, enjoyed great favor up to the most recent period, and in course of time the indications were surrounded with a formal ritualistic observance. The "bilious and gastric symptoms" were to be thus lightened; but with signs of "inflammatory hyperæmia of the stomach and liver," it was feared that emetics would increase the stasis, and with violent cerebral congestion and meningitic symptoms (not to be confounded with delirium biliosum!) they could be employed after the cerebral congestion had been controlled by venesection (Canstatt).

Diaphoretics were long used (Wilkinson), but, as well as the administration of oil of turpentine (Cox, Copland, Hinckes Bird, and others) and of iodide of potassium, again given up.

Among other specific remedies colchicum was formerly recommended by Bullock and Copland, aconite by Ringer (Lancet, 1869. I. 2), and veratrum viride by Labbé for its special action upon the pulse. Löbel and Billroth have also made many experiments with the tincture of veratrum, but they proved it to be useless in spite of the retardation of the pulse.

The chloride of iron, especially in the form of the ethereal tincture[1] (Bestuscheff's tincture), has been much praised by English physicians; it was introduced into practice by Hamilton Bell, in 1851, and has attained since then almost the reputation of a specific. According to Balfour, Campbell, and others, it shortens the duration of the disease materially, reducing it on the average from eight or ten to from two to four days, and lessens the severity of the case, that is, the typhoid and pyæmic symptoms. The remedy is always given in large doses, from a drachm and a half of the Bestuscheff's tincture to two drachms a day, and in the severest forms in from three to four times as great quantities.

---

[1] Chloride of iron in solution, in a mixture of one part of ether and three parts of alcohol. It contains one per cent of iron.—*Germ. Ph.*

Of late, quinine has been used in large doses, especially in Germany, in consequence of the well-known investigations of Binz. Liebermeister administers it in doses up to four and a half grains every two hours, and commends its influence in reducing the fever and shortening the affection. Russell, and Blake also, as well as Binz, observed a long apyrexia in some cases after large doses of quinine.

Volkmann uses cold baths to reduce the temperature, which, repeated according to circumstances three or four times a day, lessen the fever materially, and are willingly taken by the patient. They have no influence over the local process.

Opinions concerning the necessity of *local treatment* in erysipelas have differed widely at various times. Many of the older physicians especially regarded the rash as a noli me tangere, and were horrified at the use of any remedy by which the process in the skin could possibly be disturbed and the "recession" of the rash effected. Local treatment was often merely expectant, and partly on this account, partly also on account of the uselessness of many of the remedies early recommended, limited to sprinkling the affected parts with rice or bean meal, lycopodium, and similar substances, and enveloping them in wadding. Elevation of the parts affected with erysipelas, formerly recommended as being as beneficial for the diminution of the blood-flow as in phlegmonous inflammations, exerts no influence upon the course of the process, according to the observations of Estlander.

Attempts at local treatment, however, have been very numerous; individual views with regard to the nature of the disease leading to the trial of various systems.

The older physicians placed antiphlogistics in the foreground; abstraction of blood holding the first place among the means recommended for this purpose, as in general treatment. Lassis, Dobson, Bright, and others made a number of little punctures or incisions over the erysipelatous surface (from ten to a hundred or more), repeated them often, and favored the after-bleeding by a sponge soaked in warm water; Hutchinson, McDowel, and Lawrence preferred deep incisions; while Lisfranc placed from twenty to fifty leeches upon the inflamed part, because smaller numbers did not sufficiently empty the vessels, but acted rather as an irritant.

Reuss and Creutzer applied cold evaporating lotions, to which Gouzée added corn-brandy, while Rust gives the preference to warm cataplasms. Martin Solon used merely inunctions of fat; Dean, Ricord, and others, mercurial ointment instead; and Schott, lotions of corrosive sublimate (two grains to the ounce). Griscom employed glycerine, because it abstracts water from the tissues, and by this depletory action produces an antiphlogistic effect upon the inflamed organs.

As *astringents*, Velpeau used sulphate of iron in solution or in the form of an ointment; Betz, lime-water with oil; others, alum and tannin.

The derivation of erysipelatous cutaneous inflammation by blisters, has been attempted by Petit, Patissier, and others. In case it extended, it was pursued with blisters (Conté, Amiel). The remedy was employed also in order to reproduce the retrogressive exanthem upon the skin.

Under the name of the ectrotic method, Higginbottom introduced the topical use of nitrate of silver. The portion of skin affected with erysipelas, and a border of normal skin outside of it, after being freed from fat by a solution of potash, were thoroughly cauterized with the lunar caustic in substance or in strong solution. The dermatitis produced by it, according to the observations of Wernher and Volkmann, is often followed without doubt by a fall of temperature, for several hours, and often, but by no means in all cases, the farther spread of the affection is checked. Thorough painting with the tincture of iodine, recommended by Nunnely and Hasse, works in the same ray. Estlander praises the simultaneous use of subcutaneous injections of morphine and the painting with iodine, and Schwalbe, local faradization.

To prevent the further progress of erysipelas, Larrey drew a deeply cauterized line close to its border, with the actual cautery, while Fenger, Wutzer, and others, used nitrate of silver in the same way. The latter method was in favor for a long time. Velpeau employed tight bandaging, especially in erysipelas of the extremities, and Spengler painted the parts with collodium or a solution of gutta percha.

Cold, in the form of water or ice applications, is often used by Hebra and others.

Specific remedies to destroy the infectious matter *in sitû*, have often been sought for: thus Malgaigne and Jobert introduced the use of camphor externally; James and Küchenmeister, spirituous inunctions; Green, the vapors of sulphur; Schedel, chlorinated lime in solution; Kentish and Meigs, a mixture of basilicon ointment with oil of turpentine. Much more effective results are ascribed to the applications of rectified oil of turpentine, introduced by Copland. Lücke also has ecommended its application most urgently on account of its ntiparasitic action. The result in six cases was surprising, and ach time after its use there was a sensible fall of temperature. he patients often experienced a temporary burning, without he appearance of any other untoward effect. The erysipelas ften appeared to be checked, and to run its course more uickly. Borgien, Bonfigli, and others, confirm the value of his method, which has been rapidly introduced into practice. have myself seen many good results from it.

The same idea has lately been carried farther by Nyström nd Westerland, who have introduced the use of aseptin (more ccurately: amykosaseptin, Gahn) against erysipelatous inflammation of the skin.

This remedy is composed of boracic acid, and of a preparation of cloves, the ormer of which exercises a destructive influence upon animal, the latter upon vegeble parasitic organisms. It is weaker than carbolic acid, but is to be commended or its want of odor and the slight irritation it produces upon wounds.

Very recently carbolic acid has been used. Kaczorowski made quite a detailed trial of it. He mixed carbolic acid with oil of turpentine (one to ten), rubbed it thoroughly into the skin, and applied over it compresses of lead-water, and in severe cases ice in bladders or compresses also. Under this treatment the cutaneous redness becomes first more intense, and the formation of blisters more pronounced; but after from twenty-four to forty-eight hours he observed the disappearance of these symptoms, without recurrence.

Wilde boasts of good results from the subcutaneous use of sulpho-carbolate of soda, injecting from fifteen to thirty minims of a forty-grain solution into the erysipelatous portion of the skin. After the first use of it the succeeding evening temperature is reduced; after the second or third day the edge of the inflamed skin appears faded; and after the third or fourth day the erysipelas is reduced to a slight œdema. The remedy itself causes no local disturbances.

Hüter, who had previously used inunctions with tar, recommends the subcutaneous injection of carbolic acid. The local disturbances occasioned by it are, in fact, very slight; according to my own observations, and as Hüter states, the carbolic acid seems to exert even a local anæsthetic action. The results are much praised. When the injections are made at some distance from the affected part, the farther progress of the erysipelas is said to be arrested. Aufrecht obtained similar favorable effects upon four old persons—one of them eighty-two years old—in whom five injections of carbolic acid into the healthy subcutaneous cellular tissue in the neighborhood of the erysipelatous part checked the progress and effected the diminution of temperature, and a rapid improvement of the general condition. (Several trials undertaken lately in the Charité, however, yielded no such favorable result.)

According to these experiences, a specific method of treatment cannot be considered as established, although several of the remedies mentioned, especially the use of oil of turpentine, the subcutaneous injections of carbolic acid and its preparations, and the administration of the muriated tincture of iron, as by English physicians, are worthy of farther trial. What we would at present designate as rational treatment is the following:

The expectant treatment should be preferred. Mild cases scarcely demand any treatment besides rest in bed and keeping cool. In cachectic persons, or in those suffering from other serious diseases, a stimulating method from the beginning, the administration of a nourishing diet, wine, beer, or other alcoholic remedies, is not to be neglected. Weakness of the heart's action, which is always to be guarded against in extensive dermatitis, because it easily leads to collapse, demands stronger stimulants.

In more serious forms, with high fever, the mineral acids may be used, and quinine in large doses (from three to five grains several times daily), according to the method of Binz and Liebermeister. As a very valuable means of reducing the temperature, especially in protracted cases, with remissions, cold baths, repeated several times daily, are to be employed. They would be contraindicated only by great weakness of the heart's action. Fear of metastasis may be regarded as antiquated.

When there are violent cerebral symptoms, prolonged cold pplications to the head, or the douche, are in most cases suficient. Local depletions by leeches, which many physicians elieve should not be omitted, are of only temporary advanage; the treatment is better assisted by active purgatives.

Severe gastric symptoms are sufficiently relieved by mild athartics (calomel or neutral salts). Rarely is it necessary to se emetics, according to the fashion of the older physicians.

Cases where an affection of the larynx, and especially œdema f the glottis, are to be feared, demand especial attention. We ust try to relieve threatening symptoms by the application and nhalation of astringent remedies, such as tannin, alum, and the ke; and, when necessary, proceed to tracheotomy, with subequent continuation of this treatment. The energetic use of old, by ice bags and the administration of small pieces of ice, hich the patient may take in the mouth at short intervals, is lso to be strongly recommended.

Local treatment may be limited to sprinkling with powered starch or similar substances, covering with wadding, and keeping the affected part still. A daily painting with collodion, several times repeated, to which glycerine (one part to fifteen) may be added to give elasticity and prevent cracking, gives a good covering, which at the same time exerts a mild compression. In very violent inflammation the local use of cold (ice-bags and the like) is indicated.

Great tension of the skin, of especial importance in places threatened with gangrene, is materially relieved by superficial punctures, and also by warm poultices. Gangrene of the skin, especially of the eyelids, and abscesses require the proper local treatment.

MILIARY FEVER.

DER SCHWEISSFRIESEL.

(FEBRIS MILIARIS, FRIESEL, SUETTE MILIAIRE.)

The extensive older literature of miliary fever can only be used after the critical winnowing it has undergone in the following works:—*Hecker*, Der engl. Schweiss. Berl., 1834.—*Seitz*, Der Friesel. Eine histor. path. Unters. Erlangen, 1845.—*Foucart*, De la suette miliaire. Paris, 1854.—*Hirsch*, Virchow's Arch. VIII. u. IX.;—Handb. der histor. geogr. Pathol. 1860. I. 256;—From *Hecker's* grossen Volkskrankh. d. Mittelalters. Berl., 1865. (A searching critique with a comprehensive use of literature.) Among later works the following are to be mentioned:—*Galtier*, Rapport sur l'épid. de suette miliaire qui a régné dans l'arrond. de Castelnaudary (Aude) pend. l'année 1864. Toulouse, 1866.—*Dumas*, Hist. d'une épid. de suette miliaire qui a régné pend. les mois de Mars, Avril et Mai, 1860 à Draguignan (Var.) Montpell., 1866.—*Putegnat*, Journ. de méd. de Brux. Janv., 1866. *Ottoni*, Gaz. med., Lombard. Nr. 18, 19. 1866.—*Bastard*, Étude sur le trait. de suette miliaire. Avantage des bains tièdes. Paris, 1867.—*Gresser*, De la curabilité const. de la suette, dite miliaire, ainsi que des affections qu'elle complique. Paris, 1867.—*Coural*, Hist. de la suette miliaire qui a régné à St. Chinian 1865–1866. Montpell. méd. 1867 and 1868.—*Plouviez*, Essai sur la suette miliaire. Paris, 1868. (Épidemie en Pernes-en-Artois, Dpt. Pas-de-Calais.)—*Ferber*, Arch. d. Heilk. 1869. S. 335. (Description of a sporadic case.)—*Teilhol*, Étude sur la suette miliaire. Thèse. Paris, 1869.—*Bernard*, Annal. de la Soc. de méd. d'Anvers. Jan. and Fevr. 1869. (Epidemic in the Arrond. Beziers.)—*Nolé*, Journ. des connaiss. méd.-chir. Nr. 11. 1870.—*Santini*, La migliare esaminata nelle sue pertinenze morb. Firenze, 1870.—*Gresser* (*Poitiers*), Bull. de l'Acad. de Méd. XXXV. 569. 1870. (Recommendation of quinine.)—*Liverani*, L'Ippocratico 1871 (in 16 Numbers; Epidemic in Fusignano; demonstrates, in opposition to *Hebra*, the independence of the affection).—*Barbieri*, L'Ippocr. 1872. (A comparative delineation of typhoid fever and miliary fever in support of the essential nature of the latter. A disputation upon this subject by the same, *Galletti* and *Guizzardi*, ib.

In the course of time, especially during the last century, iseases of the most various kinds, which were accompanied by perspiration and showed a tendency to the formation of miliary vesicles, came to be called miliaria. A puerperal miliaria, a rheumatic miliaria, and the like, were spoken of. The specific type of the disease, which was first made known to us by the severe epidemics of the "Sudor anglicus," and from which the title was derived, retreated so far into the background that not a few physicians arrived at the theory that there was in reality no essential disease of this name. Even in the most recent times there have been discussions upon this point. In the meantime, historical and critical investigations have succeeded in again separating the different diseases improperly called miliaria from the idiopathic affection.

By the name miliaria ("Friesel," or, better, Schweissfriesel, according to Hirsch), under the revival of the old term, is meant a specific disease which occurs in the form of circumscribed local epidemics, and extends less frequently over large districts, appearing, when it does so, nearly simultaneously in various places. The epidemics come at indefinite periods, in times of moist, warm weather, and last, as a rule, only a little while, generally one or more weeks.

The disease is febrile, seldom begins suddenly, but generally after two or three days of prodromata, and runs its course more or less regularly in two nearly typical stages. The first is characterized by a profuse sweating, which lasts one or two days, and by a very harassing feeling of compression at the epigastrium, by præcordial anguish, and violent palpitation. After the sweating there appears in the second stage a rash like that of measles, the spots of which exhibit in their centres miliaria vesicles. This lasts two or three days, and ends in extensive desquamation. The remaining symptoms are less characteristic, consisting mostly of constipation, loss of appetite, headache, more or less uneasiness, and pains in the limbs. In many epidemics, diarrhœa and bronchitis are frequent complications, in others they are seldom observed.

The disease runs its course, according to time and circumstances, with varying severity. In many epidemics the mortality is very slight, while in others it rises as high as twenty per cent. or more. Death is generally preceded by an increase of the nervous symptoms, and does not reveal any specific anatomical lesions. Convalescence is generally much protracted, because the patients are considerably emaciated and debilitated.

HISTORY AND EPIDEMIOLOGY.

The history of miliaria extends only as far back as the fifteenth and sixteenth centuries. We are acquainted with a widely spread epidemic of this period, known by the name of the English sweating-sickness, which, according to the investigations of Hecker and Hirsch, is identical with miliaria, or at least approaches it very nearly.

The English sweating-sickness is described as an extremely violent fever, which began after a short chill, with cardialgia, headache, and lethargic stupor, destroyed the strength at the onset, and ended within twenty-four hours. According to the excellent description of Hirsch, in his edition of Hecker's "Great Epidemics of the Middle Ages," palpitation and anxiety, excessive perspiration, and rheumatic pain in the neck, were its characteristic symptoms. In many cases there were extensive eruptions upon the skin, accompanied often by vesicles or nodules. The irritability of the skin and the tendency to dangerous metastases were so great that the patients were unable to change their linen during the sweating without fatal consequences. The mortality of many epidemics was frightful; eighty to ninety per cent., and often more, of those affected died, while other epidemics ran their course with remarkable mildness.

The disease was first disseminated in England, after the battle at Bosworth, by the army of the victorious Henry, on the 22d of August, 1486, a year distinguished by extremely wet weather. Immediately after his ceremonious entrance into London, the disease began to rage fearfully in the densely inhabited streets. Two mayors and six aldermen died within a week, almost before they had laid aside their festal garments. Many, who were rejoicing at evening, in the morning were no more among the living. The pestilence chose most of its victims among the strong men, and as the heads of the most renowned families and of the great mercantile houses were lost, together with those who were the support of countless people of more humble station, the gayety of the celebration was turned into the deepest sorrow.

Recovery from the disease gave no security, as many of those who had survived one attack were affected with the same severity for the second and third time, so that even the small consolation of plague and small-pox patients, that of going through life, after their escape from death, free and unconcerned, was taken from them. Thus the pestilence spread until the end of the year over all England, and raged in all places with the same violence as in the capital.

The physicians, being exclusively Galenites, knew not what to advise. The people, thrown upon their own resources, fell back upon old and well-known English methods of treatment: no powerful drugs, but a moderate temperature; no nourishment, but a little mild drink, and quietude for twenty-four hours, until the issue was decided.

The next epidemic in England occurred with much less violence in the rainy summer of 1507, and lasted only until autumn. The succeeding one appeared in July, 1518, lasted six months, and showed even greater activity than the first, in 1486. It ran so rapid a course that the patients were snatched away in two or three hours, and the first chill was regarded as the announcement of certain death. Among the lower ranks the number of deaths was countless, but the nobility also were not spared; no precaution availed to keep death from their palaces. Many persons of the king's household died, and in many places one-third or one-half of the inhabitants were taken off.

The disease appeared for the fourth time, and with equal intensity, in England in May, 1529, a season of exceptionally abundant rains and mists. Historical writers spoke for a long time of its excessive mortality. In July of the same year it appeared for the first time in Hamburg, introduced, as it was supposed, by a ship-captain (Hermann Evers) arrived from England. Twelve of his crew and passengers died within two days after their arrival. The extension of the pestilence took place with great rapidity, 1,100 of the citizens dying within twenty-two days. A short time afterwards, or almost simultaneously, it showed itself in neighboring cities, and also in Zwickau, fifty miles distant, without previously touching Leipzig. It soon occupied an immense region, extending on one side as far as Dantzic, on the other to Strassbourg, and southwards to Vienna. It appeared, however, that "the flames did not start from one focus, but were kindled, as if spontaneously, everywhere, and were met with in all places." Denmark, Norway, Sweden, and perhaps also Poland and Russia, were attacked. It was remarkable that the Netherlands, in spite of their close connection with England by navigation, were visited four weeks later than Hamburg, and that the first cases appeared, as was observed in England at the time of the epidemics, while a thick mist lay over Amsterdam.

It was at this period that the custom was introduced into its treatment of allowing patients to sweat twenty-four hours uninterruptedly, to aid the critical excretions. They were placed immediately in bed, and covered with feather-beds and furs; the rooms were highly heated and the doors and windows carefully closed. A single physician in Zwickau only, whose name is not preserved, vigorously opposed "this fatal error." He went from house to house, and wherever he found a patient buried in hot beds, he tore them off with his own hand, and forbade them from murdering the patients by heat. By his determined bearing he rescued many who must otherwise have been smothered.

The last outbreak of the disease in England was in 1551, and was unchanged in violence. It appeared again in spring, also at the time of an intense fog.

After this the disease was no longer observed in this wide-spread distribution and intensity; a circumscribed outbreak only, the sweating-fever of Röttingen (Sinner), which occurred nearly 250 years later, in Nov. 1802, during rainy and misty weather, recalls those descriptions. The epidemic was limited to this town, and ran its course in ten days, but caused frightful mortality. It was characterized by a pouring sweat, tearing pains in the neck, and palpitation, and led to death or recovery within twenty-four hours.

If we review the epidemics thus found recorded in history, their resemblance to miliaria in etiological and symptomatic relations leads to the theory, according to Hirsch, that they represent a morbid process, if not identical with it, at least differing from it only by gradual modifications. The sudden occurrence and the excessively short duration of the epidemics, oftentimes, their dependence upon certain seasons and conditions of the weather, the peculiarity of running a highly fatal course at one time, and a mild one at another, are common to both. In both the outbreak occurs at night, with chills, prostration, pains in the limbs and back, anxiety and palpitation, profuse sweating, and a rash, which latter, however, is not constant. Moreover, the same course, and like disturbances of convalescence are common to both. Finally, the history of the Röttingen epidemic establishes the connection of both diseases in so striking a manner, that it can be correctly ranked with the latest malignant epidemics of miliaria as well as with the mediæval form of the disease.

In the further history of miliaria there have accumulated so many errors and misinterpretations, that only the most persevering critic, as Hirsch has shown himself to be, could succeed in following the historical record of the disease in its epidemic outbreaks.

Men had gradually begun to disbelieve in the essential nature of the disease, and to attach undue importance to certain symptoms; at one time to lay special weight upon the red papular rash (" purpura "), at another upon the occurrence of sweating accompanied by vesicles (" miliaria "). Thus it came to pass that, by degrees, the most different forms of disease, in the course of which " purpura" or " miliary vesicles " appeared, were called " miliary fever." The peculiarity of the affection was in this way so hidden, that not a few physicians, and especially dermatologists like Hebra and his school, came to deny the existence of a miliary fever, and to refer the belief concerning it to a vesicular eruption which occurs as an accidental complication due to excessive sweating in the most various febrile affections or artificially produced. Historical investigations have furnished, however, in contradiction of these negative views, proof of the existence of the contested specific affection.

After the epidemics of the sudor anglicus the disease disappears completely for more than 160 years. We meet it again, according to the historical evidence, which has lately been carefully sifted, only since the beginning of the last century, and especially in France and Italy, and likewise also in Germany, Austria, and Belgium. The first epidemic, best known under the name of "the sweating sickness of Picardy," appeared in the year 1718 (perhaps even somewhat earlier) in different districts of Picardy and the neighboring provinces. After this (compare the tables of Hirsch) followed 174 epidemics up to the year 1861, and since then an additional and not inconsiderable number on French territory, which are described by Dumas, Galtier, Coural, Plonviez, Nolé, and others. They occurred at varying intervals and in very various localities, always confined, however, in point of time and space, within narrow limits. The area most particularly affected has been a portion of territory in the north-east of France, which includes Normandy, Isle de France, Picardy, Flanders, the northern portion of Champagne, and Franche-Comté, and comprises also a part of Elsass-Lothringen. Other districts, especially to the southward, have been less frequently visited.

Italy, too, has been attacked with quite a long succession of epidemics.

After the disease had prevailed in and around Turin from 1715 to 1720, there appeared the following outbreaks in other districts of Piedmont: in Modena in 1775; in the Venetian provinces in 1790 (at Modena, and later in other places); on the plains of Lombardy at the beginning of this century; at Milan 1844, 1846 to 1848, and at other times; in Tuscany (Florence) in 1836 and 1837. Since then, both there as well as in other regions of upper and middle Italy, several epidemics have been observed. The most recent of these have been described by Ottoni, Santini, and in a particularly thorough and critical manner by Liverani.

While middle and north Germany, contrary to former views, show in all, since 1801, only four small isolated epidemics (Wittenberg, 1801, district Kalau in the autumn of 1838, Frauenstein in Saxony in the winter of 1839, Wegeleben in the winter of 1849), there have been observed several larger ones especially in the south-western parts, in Würtemberg, Bavaria, and Baden. Single epidemics have appeared also in several of the mountainous districts of upper Austria, Styria, and Galicia; also in Belgium, in 1849 (together with the cholera), at Lüttich, Namur, and in the vicinity of Mons, and in 1850 in Luxembourg (Hotton).

Ferber describes an affection observed in a patient from Caracas, which he considered "idiopathic miliary fever." It is doubtful, however, if sporadic cases occur in this way independently of epidemics. The example is not free from objection. Nine single cases of remitting fever, with the formation of phlyctenæ, also observed by Wunderlich,[1] do not belong here.

Historical research shows that miliary fever has been confined in its geographical distribution within more narrow limits than almost any other infectious disease. Its epidemics are often limited to single places, or spread only over definite districts. Only exceptionally does the disease show a wider spread over an extensive country, and then it is often found that the fever has arisen at the same time in different places. It seldom spreads by gradual progression, but more often by leaps over broader districts.

The period of duration of the epidemics is also limited. They last on the average only from seven to fourteen days, seldom two or three months; and then generally run their course in such a way that the majority of the cases are confined to the first two, three, or four weeks, and the further prolongation of the epidemic is shown only by single cases.

On the other hand, the epidemics often attain in some places that are affected a very wide prevalence. In one which spread over a portion of Languedoc in 1782 (Pujol), more than 30,000 persons were affected; and in the epidemic of Forcalquier, in 1772, out of 2,000 inhabitants 1,400 were attacked. On the average, according to Hirsch, from ten to twenty per cent. of the inhabitants are affected; it rises even to thirty per cent. or more; epidemics are not rare, however, in which the number is much smaller.

ETIOLOGY.

The epidemics of miliary fever show, on the whole, a very decided dependence upon the seasons of the year. Of the 174 mentioned by Hirsch, fifty-nine began in spring, seventy-eight in summer, but only eight in autumn, and twenty-eight in winter. Five-sixths of all the epidemics prevailed in spring and summer, while the disease was extremely rare in autumn, and although somewhat more frequent in winter, yet it occurred only in very limited outbreaks. (The epidemics of the English sweating-sickness also began in spring and summer.)

---

[1] Arch. d. Heilk. VIII. 174.

In view of such a decidedly expressed preference for particular seasons, we are authorized in seeking the cause of the disease in the atmospheric conditions peculiar to these seasons. The great majority of cases shows, in fact, that the disease has developed with predominant frequency in warm and moist or very changeable weather, or directly after it. At the time of most of the winter epidemics, too, which have been of two or three weeks' duration only, a "moist, dull" weather was observed. Moreover, in the history of the English affection, the coincidence of its occurrence with the appearance of misty and damp weather was so pronounced that Hecker was inclined to recognize in it a relationship of cause and effect.

On the other hand, it cannot be demonstrated, according to Hirsch, that the occurrence of the disease is dependent upon the condition of the soil.

From the observations concerning its prevalence during damp weather, many physicians were inclined to connect its appearance with the boggy and moist condition of the ground. It had been noticed that the first epidemic in Picardy spread along a moist valley in a peat district, but that it spared a chalk district of the neighboring plain. Other epidemics led to the same observation; in those of 1772–73 in Provence, of 1812 in Elsass, and of 1820 and 1824 in Bavaria, the disease was confined to deep, moist valleys; and that of 1829, in Ensingen, was preceded by an overflow. Barthez mentions that the epidemic of 1839, in Canton Rebais, affected only the valleys which were exposed to frequent inundations; and Martin Solon and others state that that of 1841, in Charente, was confined to the swampy shore of the Lione, and extended farther into the country with diminishing intensity. In opposition to these and numerous other observations, it has been shown that not a few epidemics have developed upon dry, airy, and elevated plains, avoiding swampy and low valleys, as, for instance, in the years 1810, 1821, and 1832, in the Department of the Oise, in 1820 in Giengen, in 1830 in Mettingen and Gmünd, and in numerous other cases. In the epidemic of 1866, in Pernes-en-Artois, three airy and elevated places, and three lying in the valley, were alike affected. Similar relations are stated in the descriptions of several of the latest epidemics (Teilhol, Liverani, and others).

Finally, we have, in opposition to the theory that the poison is really promoted by a swampy region, the evidence that the extensive swamps of Gascogne were ly attacked by the epidemics after 1840, and only slightly at that; while in per Italy the first epidemics were confined for a long time to elevated places.

Nevertheless many epidemics do show a connection with cerin contaminations of the soil. In that of St. Chinian, in 1864–, Coural, for instance, made the observation that the neglected ndition of the drains, and the collections of refuse in gardens, d the like, if they did not create, at least contributed to the rther spread and severer form of the disease. It seems to him pecially deserving notice, that when a canal situated at one d of the city was cleaned in April, 1866, and the mud from it as allowed to lie upon the shore, all the houses in the neighrhood were visited within five weeks by the disease, and in its verest forms, while in all other parts of the town only mild ses occurred. Similar observations were made in the epimic of Noyons in 1849, and in Languedoc on the Canal du idi.

As regards individual predisposition, it appears that the disse affects on the average more women than men, and espeally the vigorous age between twenty and fifty years. The verse relation is relatively infrequent.

In the epidemic of 1821, in the Department of the Oise, there were 803 male d 1,177 female patients; 1,461 were between the ages of sixteen and fifty, while 56 were below, and 284 were above those limits. In the epidemic of 1851 in rentan, 97 men and 181 women were attacked; among them were 19 children. that of 1854 only women were affected. The epidemic of 1844, in Bavaria, tacked 2,109 women and only 1,535 men. In single epidemics, on the other hand, at for instance of 1845, in Poitiers, and of 1855 in Hastinguer, the numbers in th sexes were nearly alike, and occasionally the male sex was in excess, as in 1849 Niort and Dôle, in 1854 in Marvejols, 1865–66 in St. Chinian (among 107 only 5 women), 1866 in Pernes-en-Artois (40 per cent. women), and in Davayat (Puy-deôme).

The disease appears especially to attack healthy and strong ersons. The puerperal condition, typhus, and other diseases in o way produce a special predisposition to it, as was formerly elieved.

From the first appearance of the English sweating-sickness, it as been shown that the disease affects all classes of the people like, and is in no way confined to the poorer. This peculiarity has been confirmed by later observations. It has been further noticed, moreover, in this connection, that the spread of the disease is not promoted even by the collection of great numbers of persons in institutions of various kinds, as prisons, barracks, and the like; sometimes such establishments are even wholly spared.

"Observation has shown," writes Parrot, whom I quote as an example (Epidemic of 1841 in the Dordogne), "that the cases diminished in numbers and severity the greater the aggregation of individuals; . . . . in Périgeux all the establishments in which great numbers of people dwelt together were spared. In the barracks, where two battalions were generally quartered, as in the college, where there was no vacation during the first days of the epidemic, not a case occurred, and in the prisons, containing from 100 to 120 inmates, only three were slightly affected."

Concerning the *exciting causes* of the disease we know very little. In the earlier epidemics (Loreau in Poitou, 1845; Rayer, 1821, in the Department of the Oise, and others) it was believed to have been noticed that the disease could be communicated by intercourse. Fourcart, however, in his observations of the epidemic in the Department of Somme, 1849, came to no definite conclusion concerning this, but considers a "transmission infectieuse," in the same manner as it is accepted in the acute exanthems, as probable. At all events, miliary fever is not contagious in the sense that the disease may be conveyed directly from the sick to the well. The limitation of many epidemics exclusively to a single place is opposed to such a theory, and, moreover, they do not spread in establishments where great numbers of people live together. Finally, the disease may attack certain individuals among the families and inmates of a house, while it spares the others.

Attempts at inoculation with contents of the vesicles have been without result, although Seitz regards the fluid as the vehicle of the contagium.

The occurrence of miliary fever simultaneously with epidemics of scarlet fever, and with those of measles, has been observed (1820 in Giengen, 1830 in Gmünd, 1831 in Esslingen, 1855 in the Department of Jura). The cases are, however, so infrequent that they do not support the repeatedly expressed suspicion of a relationship between the poisons.

h regard to cholera, a certain relation between it and miliary fever has been ed by some writers. In the earliest cholera epidemics, Hufeland called atten- their resemblance to the Sudor britannicus. It is peculiar that subsequently l cholera-epidemics were accompanied by or followed miliary fever (1832 in epartment of the Oise, 1849 in the same and neighboring departments, 1854– several of the middle and southern regions of France, and elsewhere). Some- s, also, the disease has appeared in the wake of cholera (1849 in Tournay and where). Very remarkable individual observations have been made, showing that iary fever and cholera may spread in neighboring districts, and appear to lude each other; in La Marche, for example, with 2,000 inhabitants, there were, 1854, ninety-seven cases of miliary fever and only forty-three of cholera, while in ameraus, near by, the reverse proportion obtained.

Finally, cases are described in which, with various modifications, one and the me person is attacked by both affections simultaneously, or one after the other. n accession of miliary fever is supposed to exercise a favorable influence on the ourse of cholera (Verneuil); the reversed relation, however, is thought to hasten the nfavorable result.

It is difficult to form a definite judgment as to these relations. Although not very numerous, the observations appear to be satisfactorily founded. Hirsch is inclined not to regard the coincidence of the two diseases as accidental, and reminds us that the affection described by Murray under the name "sweating-sickness," during the Indian epidemic of 1839–40, appeared to afford a certain protection against the cholera.

A peculiar sweating disease was also observed by the naval surgeon Roux,[1] which occurred at Toulon in the cholera years 1849, 1854, and 1855, and was essentially characterized by profuse intermittent sweats, with great weakness, but ending favorably. A similar affection, with a very malignant course, and with symptoms of asphyxial cholera, occurred in the French fleet in the Mediterranean during the Crimean war, and also (according to the statements of Houle and Bourgogne) in some of the French Departments during the cholera epidemic of 1854; in place of the intestinal discharges, however, there was excessive perspiration.

A relationship of the two processes cannot of course be deduced from this evidence, and least of all are we warranted in regarding cholera, as Dubun does, a sort of "internal miliary fever" (Suette interne), or the latter as a kind of "skin-cholera." The facts hitherto noticed, however, demand that this relationship should be more thoroughly observed in future.

Anatomical Changes.

Reports of dissections in miliary fever are not very numerous, and reveal no characteristic changes, at least not such as suffice to explain the violent symptoms during life.

---

[1] Union Méd. 1855, Nr. 27 et seq. 1857, Nr. 131 et seq.

The most striking is the rapid occurrence of decomposition, which, as Galy[1] says, "begins almost during life. A few hours after death the skin is everywhere œdematous, frothy blood flows from the nose and mouth, and the odor of decomposition quickly prevails."

The internal organs show nothing positive except great hyperæmia. The lungs are much congested, and the mucous membrane of the bronchi and trachea is found reddened and often covered with reddish mucus. The heart is soft (Galy and others), and the pericardium occasionally ecchymosed (Primbs, Borchard). The mucous membrane of the stomach and intestines appears generally reddened (Rayer, Dubun), occasionally studded with red spots (Primbs), and, in the small intestine, with "vesicles," which some (Barthez, Landouzy, and others) regard as swollen solitary follicles; others, as Bourgeois, as distinct miliaria vesicles formed of epithelium and filled with fluid, which occur here as the analogues of the cutaneous eruption.[2] Immediately over the valves superficial follicular ulcers are sometimes found (Beck). The liver is full of blood, and somewhat soft, and the spleen is always enlarged, softened, and often friable (Borchard and others).[3] Galy found the kidneys generally hyperæmic, Primbs describes them as normal.

In the central organs of the nervous system nothing abnormal is found except occasional hyperæmia and a more or less considerable œdema of the meninges (Liverani). Theden[4] alone has several times seen the sheaths of some of the cervical nerves (the fifth and seventh pairs), as well as the ganglia of the cervical sympathetic, filled "with yellow serum."

The blood in the dead body is uniformly described as thin and dark-colored, while that drawn during life by venesection is bright red, thin, and coagulates imperfectly.

The contents of the miliaria vesicles have been examined by Seitz and Beroaldi.

---

[1] Canstatt's Jahresber. II. 14.

[2] *Eller* describes similar vesicles upon the liver, *Seitz* also upon the pericardium and on other parts.

[3] *Hirsch* calls attention to the fact that this is found mostly in persons who have lived in malarial regions.

[4] Compare Canstatt's Handb. 1874, p. 218.

It is clear in the beginning, and contains, besides many smaller nuclei, cells which are smaller than pus-corpuscles (!), with three or more nuclei, which remain visible after the disappearance of the cell membrane by the addition of acetic acid. Seitz seeks in these nuclei and cells, which he observed also in the lymph of varicella and cow-pox (without doubt only pus-corpuscles), the real virus of the disease. In the later stages of the vesicles, these cells appear in greater quantity, until they finally form nearly the whole contents of the oldest and dried-up vesicles.

Symptomatology.

A prodromal stage of two or three days' duration precedes the utbreak of the disease in cases of moderate severity. The atients complain of great irritation of the skin, dryness of the nouth, increased thirst, weakness, headache, and feebleness of he limbs. There is often a peculiar painful sense of oppression n the region of the stomach, which not unfrequently increases to n intense feeling of suffocation peculiar to the disease (Nolé and thers). There are occasionally also ringing in the ears and dizziness. In milder cases these precursors are wanting (Coural).

The beginning of the disease generally occurs, according to Plouviez and others, as well as the older observers, in the night, r late in the afternoon or evening, and, as a rule, it is indicated nly by a slight chilliness, seldom by a pronounced chill, and is haracterized by a profuse and persistent sweating. Its onset is ccompanied by a feeling of prickling and stinging in the skin, ither first upon the head and breast, gradually descending, or imultaneously over the whole body, and is so abundant that it uickly penetrates bedding, mattresses, straw-beds, and everyhing, and often macerates the skin, as it were. The sweat is of cid reaction,[1] according to Seitz, and diffuses an unpleasant dor, which has recently been noticed by Plouviez also, but hich was referred by earlier observers (Foucart and others) to apid decomposition promoted by uncleanliness.

At the same time a more or less severe fever sets in, the pulse s quickened to over 130 in the minute, and the skin is burning ot. The headache increases, and in many cases (in one-quarter f the patients, according to Plouviez) the patients experience, together with a violent and tumultuous palpitation and abdominal pulsation, a feeling of constriction in the chest and the epigastrium (barre épigastrique), and precordial pain. These symptoms increase not infrequently to a frightful degree, although neither in the heart nor in the lungs is any anatomical lesion to be discovered. The respiration becomes at times irregular and interrupted. The epigastrium is extremely sensitive and painful on pressure.

---

[1] *Stahl* did not find it always acid, and *Barthez*, who examined only that of the face, ound it neutral.

This condition remains unchanged until the outbreak of the rash, or it undergoes exacerbations, occurring irregularly or sometimes in such regular intermissions that the suspicion has suggested itself to many observers that they were dealing with intermittent fever.

The rash appears on the third or fourth day of the disease, seldom earlier; but in other cases not until from the seventh to the tenth, or even until the fifteenth day (Coural), and is often attended with a marked increase of all the symptoms, and a stinging, pricking feeling in the skin. The other symptoms, as a rule, disappear either suddenly or gradually after its outbreak. In the milder cases, the efflorescence is often wanting (in ten per cent. of Plouviez' cases, and in one-third of Verneuil's).

The exanthem consists, as a rule, of small, round, or irregularly shaped spots, of from two to five mm. in diameter. They are sometimes so closely aggregated that the skin appears uniformly reddened (Coural), or they become confluent and look like the eruption of scarlet fever. In their centre there arise, after some hours, according to the statements of Coural and many of the older writers, vesicles, which in the beginning are so small that they are to be discovered only by passing the finger over them, or by the magnifying-glass. They soon become larger, however, and attain the size of a millet seed (hence the name miliaria), or sometimes even that of a pea. They contain a clear fluid, which gradually becomes milky and yellowish from pus (miliaria alba). Finally, after two or three days they burst, or dry up and form crusts, which are cast off in the form of scales. In milder cases the vesicles appear also without the red spots (miliaria crystallina).

The rash spreads by distinct advances, one after the other, ometimes with fever, and first appears upon the neck and breast, hen upon the back and extremities, less frequently upon the bdomen and scalp (Coural). Sometimes it occurs also upon the mucous membrane of the mouth and nose, and upon the conjunctiva. Foucart and others noticed it more frequently upon the mucous membrane of the mouth, where it easily led to excoriations.

These symptoms were observed in all the epidemics, according to the descriptions, and are therefore characteristic. The remaining symptoms are developed with varying frequency at different imes; headache, dizziness, and sleeplessness accompany the ffection quite often. There are generally also loss of appetite, nnatural taste, moderate thirst, and nausea, but seldom vomitg. Persistent constipation is the most uniform of all.

The urine is turbid, high-colored, and scanty; sometimes comlete suppression has been observed (Foucart and others), so that ossibly a certain portion of the particularly violent symptoms ay be referred to uræmia. In some cases strangury and pains n the region of the bladder and elsewhere have been noticed. edoni considers the profuse secretion of urine, which sometimes ccurs during or after the sweating stage, a favorable sign.

The disease ends with more or less extensive desquamation; onvalescence, however, in consequence of the great debility and maciation, is often protracted much longer than the severity of he disease would lead us to expect, and is sometimes delayed by regular sweats, furuncles, and the like.

This nearly typical course is not infrequently modified in varius ways.

In the severest forms (Coural and others) all the symptoms, nd especially the heat, reach a very high degree at the time of n exacerbation, and the feeling of constriction and the precorial pain are so great that the patient believes he must suffocate. ometimes they increase to a sensation of being laced together barre trachéobronchique), extending upwards from the epigasrium to the larynx, and of apnœa (Bazin, Verneuil), so that true ases of suffocation occur without perceptible changes in the eart or lungs. The patients have no rest for a moment, and hrow themselves about in bed, or fall into raging delirium. The sweating and rash are often absent, and the patients die with muscular cramps or convulsions.

Occasionally the course in the sweating stage is fulminant; the patient, up to this time comparatively easy, utters a few disconnected words, the face becomes cyanotic, the carotids and the abdominal aorta pulsate violently, and a fatal collapse quickly follows.

These cases call to mind the most violent forms of the English sweating-sickness; but they appear to have become less common in the most recent epidemics. Of seven cases of this sort recorded by Coural, five died between the second and fourth days, two in rapid collapse, three in an accession of delirium and convulsions.

There is sometimes developed in the sweating period a grave typhoidal condition, as among others Nolé describes it, which leads to death, preceded by somnolence or coma, syncope, a soot-like coating upon the teeth and tongue, and profuse bleeding from the nose and uterus, and in which no considerable anatomical changes are to be discovered.

In mild cases, on the other hand (abortive cases, according to Nolé,), the whole process is often ended in from three to five days. They run their course without prodromata, and often without the rash (suette sans éruption), and only with repeated sweatings, sometimes so slight that the patients do not need to give up their business.

Complications are not common. Sometimes an angina accompanies the affection. A more or less intense bronchitis is especially mentioned. Pneumonia was seen only once by Coural, and Robert believes that pneumonia is sometimes thought to be present when auscultation shows nothing abnormal. In some epidemics, especially perhaps in the winter season, bronchitic affections appear to be more common, in others (at cholera seasons?) diarrhœas.

Diagnosis.

The diagnosis offers no difficulties during the epidemic occurrence of the disease. The profuse sweatings, with prickling of the skin, the oppression and precordial pain, and the subsequent outbreak of the rash assure against any possibility of confounding this disease with measles. When a decidedly intermittent type prevails, the occurrence of the exanthem is generally enough to distinguish it from intermittent fever. From typhoid fever in its early stages, the disease is distinguished by the short course, the low degree of fever, the initial sweating, and the constipation.

The *duration* of the affection amounts on the average to six or eight days, of which one, two, or three belong to the sweating stage, and three or four to that of the exanthem. A longer duration of the fever has, however, been occasionally observed, when the rash does not appear until later, or in successive outbreaks. A shorter course, like that of the English epidemics, appears to belong only to the fulminant and to the very mild cases.

Relapses are mentioned by most observers. They run their course without severe symptoms, only with repeated sweatings, and mostly favorably. In other cases they seem to be produced by errors in diet, and then not unfrequently assume a severe form.

Mortality and Prognosis.

The *mortality* differs greatly in various epidemics. Sometimes fatal cases are a great rarity, whilst the mortality, in some of the recent epidemics even, rose to twenty or thirty, or even fifty per cent. (Würzburg epidemic of 1825 and others). On an average among 16,000 cases, which have been collected partly by Hirsch, and partly from the accounts of the later epidemics, a mortality of from eight to nine per cent. occurs.

In the epidemics of 1849 (Department of the Oise), 1851 (Department of Herault), 1855 (Cognac), and among 130 cases of Gresser, not a patient died. In 1842 (Department of the Lot-et-Garonne and Dordogne) and in 1851 (Carentan) the mortality was very slight. On the other hand, it rose in some epidemics (1841 in Dordogne, and 1849 in Niort) much higher. In 1866 (Pernes-en-Artois) ten per cent., eight of Liverani's sixty-eight patients, thirty of Coural's 117, and eleven of Teilhol's forty died.

The *prognosis* depends therefore essentially upon the character of the epidemic. Coural is of the opinion that it would be well to pronounce it dubious in all severe forms. With the regular course of the disease, and with moderate severity of the symptoms, the result is favorable, while high fever, excessively profuse sweating, and a considerable increase in the feeling of constriction appear to be dangerous to life. Profuse hemorrhages, somnolence, and coma, convulsions, rapid sinking of the strength, with small pulse, violent delirium, and great dyspnœa, belong to the gravest symptoms, which generally end in death.

The fatal result is seldom observed after the eruption of the rash, and during the period of desquamation. It occurs most generally in the sweating stage, before the appearance of the rash, at the time of the exacerbation, which precedes this, and with a sudden increase of the nervous symptoms.

TREATMENT.

After the experiences of the early epidemics of the English sweating-sickness had led to the expectant treatment (the so-called old English method), the theory was again adopted, during the great epidemic in the Netherlands, that the eruption and the sweating were critical manifestations, in which the patient must be aided by diaphoretics of all kinds, and by the warmest and most careful covering. This view was maintained for a long time. Foucart even was enabled, as he communicates in a graphic description of his experiences in the epidemic of 1849, to observe its practical workings in the extremely energetic use of all sorts of diaphoretic agents. This consisted in the excessive employment of materials for producing heat—beds, furs, clothing, exclusion of air, bottles of warm water, and the like—although the epidemic occurred towards the end of May and the beginning of June!

Blood-letting, which the physicians of the last century considered to be indicated, especially where there was a feeling of suffocation and apnœa, was soon shown to be injurious, and Foucart states expressly that it generally led to an aggravation of the patient's condition, and not infrequently to a fatal result.

Among special remedies anti-spasmodics and nervines, vale-an, camphor, and, under some circumstances, opium, were ten used. The intermittent character of many cases led Pigné, arrot, Galy, and others to the use of quinine, which is more cently again recommended in large doses by Gresser, Coural, d others. It diminishes the fever, but it is uncertain whether not it shortens it, although it is certainly effective against vio-nt nervous symptoms.

To incite diuresis, Schönlein gave seltzer-water, and Sedoni ld water in great quantities, to which, in persistent delirium, resser added from forty to fifty drops of solution of chloride of n daily. Chlorine water (Herzog), corrosive sublimate (Eisen-nn), and similar drugs have been tried in the epidemics of the t thirty years. In several of the later epidemics ipecacuanha s used in the beginning of the disease, according to the ample of Foucart, as an emetic (from thirty to forty grains, three doses, at intervals of fifteen minutes); its boasted effi-cy, however, has not been confirmed.

Opinions concerning the use of cutaneous irritants (sinapisms, isters, etc.) in nervous symptoms are more unanimous, especi-y in the sensation of constriction. Lately, subcutaneous injec-ns of morphine have also been used with advantage.

Schönlein, in the Würzburg, epidemic attempted to neutralize e acid secretions by washing the skin repeatedly with a solu-n of caustic potash (twenty-five to fifty grains to the ounce). ayer used cold applications to relieve the pain at the epigas-ium, and Foderé applied ice-cold compresses during collapse. ore general warm baths were employed by Bastard.

Warning is given against the use of purgatives in large doses; d bleedings, even locally by leeches and other means, are espe-ally to be avoided.

In general, a return has been made to the expectant treatment. ost physicians limit their efforts to the use of mineral acids d lemonade, or light aromatic drinks, as mint teas, etc.

Warm baths, where possible, or washing the skin with warm ater, to which vinegar or alum has been added, appear to do od. Stimulating treatment—such as alcoholic stimulants, cam-or, and the like—seems to be indicated only in the severest cases. Most observers are unanimous in recommending quinine; while in persistent sleeplessness opium, either alone or with ether, is also advised.

Attention to good hygienic conditions remains the chief point in treatment. The patient must be kept cool and lightly covered, although protected against draught, especially during the frequent changes of the clothing wet with perspiration, and during the washings and baths.

The diet should be moderately nutritious, and during convalescence the prolonged use of tonics is indicated.

From a prophylactic point of view nothing but leaving the infected region seems to be of any avail.

DENGUE, OR DANDY FEVER.

(DENGUIS.)

he Spanish word Dengue is derived from the English Dandy, and corresponds to it.

ush, Med. Inq. and Obs. Phila., 1789—*Pezet*, Med. Repos., N. Y., New Ser., Vol. V. No. II. 1819.—*Kennedy*, Calcutt. Med. Trans., I. 371. 1824.—*Mellis*, ibid., 310.—*Twining*, ibid., II. 32. 1825.—*Mouat*, ib., II. 41.—*Waring*, North Amer. Med. and Surg. J., Apr., 1830. V. IX. 374.—*Stedmann*, Edin. Med. and Surg. J., 1828, Vol. XXX. 227.—*Lüders*, Hufel. J. LXVIII. Hft. 4. 33. 1827.—*Ruan*, Edin. Med. Chir. Trans., III., part II. 1827.—*Squaer*, Lon. Med. and Phys. J., 60, 21. 1828.—*Waterson*, Med. and Surg. J., IV. 303. 1827.—*Nicholson*, Edin. Med. and Surg. J., XXXI. 115. 1829.—*Furlonge*, ib., XXXIII. 50. 1830.—*Moreau*, Rev. Méd. 1828. III. 475.—*Maxwell*, Edin. Med. and Surg. J., LII. 151 and 154. 1839.—*Lehmann*, Am. J. Med. Sci., Aug., 1828. 477.—*Hays*, ibid., Nov., 1828. 233.—*Tuite*, N. Y. Med. and Phys. J., VII. 375. 1828.—*Osgood*, Boston Med. and Surg. J., I. No. 36, 361. 1828.—*Lawson*, in Forry, The Climate of the U. S., etc. N. Y., 1842.—*Dickson*, Am. J. Med. Sci., III. 3. 1828, and IV. 62. 1829.—*Dumaresq*, Boston Med. and Surg. J., I. 32. 1829.—*Daniell*, Am. J. Med. Sci., Aug., 1829. 291.—*Raleigh*, Ind. J. of Med. Sci., New Ser., I. 452. 1830. —*Pruner*, Ib. 311. 1845.—*Lallemant*, Das gelbe Fieber etc. 91. 1846.—*Hester*, Transact. Am. Med. Assoc., II. 161. 1848.—*Fenner*, Southern Med. Reports, N. Orleans and N. Y., 1851. II. 93.—*Holt*, ibid., 437.—*Arnold*, Charleston Med. J., July, 1849.—*Dickson*, ibid., Nov., 1850.—*Campbell*, South. Med. J., Nov., 1850.—*McCraven*, Transact. Am. Med. Assoc., V. 676. 1852.—*Hearde*, ibid., 683.—*Wragg and others*, ibid., IV. 71, 173, and 211. 1851.—*Anderson*, Proceed. Med. Assoc. of Alabama, 1851.—*Smith*, Edin. Med. and Surg. J., LXXXII. 166. 1855.—*Hirsch*, Dtsch. Kl. 1852. 48, 49.—*Jarvis*, in Coolidge's U. S. Army Statist. Report, from 1839 to 1855. Washington, 1856. 365.—*Gaal*, Zeitschr. d. Wien. Aertze. 1858. No. 8.—Compare also *Hirsch*, Handb. der histor. geogr. Pathol. 1860. I. 272 et seq.—*Taly*, Arch. de Méd. nav. 1866. p. 37 (Epid. in Gorée).—*Ballot*, ibid., 1870. p. 470 (Epid. in Martinique).—*R. H. Poggio* (La Calentura roja observada en sus apariciones de los años 1865, 1867). Madrid (Epid. in Andalusia, etc.).—*Read*, Proceed. of the Sanit. Commiss. of Madras. 1871 (Epid. of Aden, Zanzibar, etc.).—*Chipperfield*, Madras Monthly J. of Med. Sci., 1872. No. 28.—*Christie*, Brit. Med. J., 1872. I. 577 (Epid. in Zanzibar).—*Vauvray*, Archiv. de Méd. nav. 1872. p. 74.—*Dunkley*, Brit. Med. J., 1872. II. 378.—*O'Connell*, Raye. Ind. Annals of Med. Sci., 1872. 137.—*Dawson*, Lancet, 1872. II. 542.—*Moodern Sheriff*, Med. Times and Gaz., Nov. 15, 1873, p. 543 (Epid. in Madras, 1872).—*Unknown Author*, Union Médicale. 104. 1873 (Epid. in India).—*J. J. de Wilde* (Niederl. Tijdschr. 1873). Dengue in Fort Willem I. in Java).—*Müller* and *Manson*, Brit. Med. J., 1873. II. 294.—*Charles*, Clinic. Lectures on Dengue. Calcutta.—*Cock*, Edin. Med. and Surg. J., XXXIII. 43. 1830.—*Aitken*, *W.*, in System of Med. Edited by J. Russell Reynolds. London, 1866. I. 258.—*Rey*, Archiv. de Méd. nav. IX. pp. 278 and 382. 1868.—*Rochard*, *J.*, Article in Nouv. Dict. de Méd. et de Chirurg. Prat. Paris, 1869. XI. 130.—*Faget*, New Orleans Med. and Surg. J., New Ser., Jan., 1874. 603.—*d'Aquin*, ibid., July, 1874. 37.—*Hoover*, Am. J. Med. Sci., Apr., 1874. p. 379.

THE name Dengue[1] is applied to an acute disease, which mostly occurs as an epidemic in hot climates, seldom sporadically, and the course of which, after a sudden onset or after slight prodromata lasting several days, consists of two paroxysms accompanied by fever, either following immediately one upon the other, or after an intermission of one, two, or three days. But the two paroxysms are essentially different one from the other; the first is characterized by continuous high fever, and numerous exceedingly painful swellings of the joints, which interfere with motion, also more rarely by an exanthem. These symptoms subside after two or three days, simultaneously with the sudden outbreak of a sweat, an epistaxis, or a diarrhœa, which is often critical.

The second febrile stage, which lasts from two to three days, is marked by a remitting fever and a more or less extensive blotchy or uniform, non-elevated, rose-red eruption, and great itching of the skin, also less frequently by swelling of the joints.

The subsequent recovery is gradual, and accompanied by decided desquamation. Its course is generally accompanied by great loss of appetite, restlessness and sleeplessness. More serious brain symptoms are absent.

---

[1] In accordance with The Nomenclature of Diseases, drawn up by a joint committee appointed by the Royal College of Physicians of London, 1869, p. 5, the name is now generally accepted.

The disease attacks all ages and both sexes; with adults it terminates favorably in recovery, and is only occasionally dangerous in children. It sometimes leaves behind long-continued and painful swellings of the joints, diarrhœas, emaciation, and great debility. Relapses also occur.

It is not yet determined whether the disease is contagious or occasioned by minsmatic influences.

HISTORY AND ETIOLOGY.

Our knowledge of the history and etiology of dengue is still but little developed, because general attention has only been directed towards it in the most recent times, on account of the greater notoriety and wide diffusion of the epidemics. The disease by preference seeks hot regions near the tropics; still, isolated cases also occur in places far removed from there (New York and others).

The reports refer chiefly to outbreaks of the disease lying far apart from one another in point of time. It is still impossible to decide whether in the intervals it is completely extinguished, and constantly springs up again indigenously at certain times (date-harvest), or whether a connection is established by means of sporadic cases, which may accidentally develop into greater epidemics.

Our first information about the disease, according to de Wilde, dates from the year 1779. The chief physician of Java at that time, David Brylon, informs us briefly of an epidemic disease under the name of *knockelkoorts* (bone fever), which attacked many of the natives and colonists in Batavia. In the next year (1780) Rush describes a dengue epidemic in Philadelphia, and at the same time the missionary (Wise according to an anonymous French writer) observed the same disease on the coasts of Coromandel, Africa, Arabia, Persia, and Thibet. After a long interval it is reported (Pezet) that it occurred in Lima in 1818. Eight years later it appeared in Savannah.

According to Hirsch the almost epidemic spread of the disease beginning in America in 1827, and which was then described under the name of break-bone fever, excited greater attention. It showed itself first in September upon the island of St. Thomas, the next month in St. Croix, went thence in one direction over the Antilles towards the mainland of North America, in the other over the Caribbean islands towards Columbia, and lasted until September, 1828, at which time it had extended to Savannah. Sporadic cases also occurred in many of the large cities of North America, such as Boston, New York, etc.

After some outbreaks which remained isolated (1831 in Louisiana, 1844 in Mobile, etc.), the fever extended in the summer of 1848 simultaneously with the yellow fever in New Orleans, Mobile, and the neighboring regions. It was popularly called Dandy-fever (Spanish, Dengue) from the stiff, affected gait the patients were forced to adopt. Some isolated cases followed in the next two years.

A new epidemic attacked the Southern States of North America in 1850, and again established itself especially in Savannah, Mobile, and New Orleans, extending further, however, into Texas. The last epidemics known to have occurred in this country were in the years 1861 and 1866.

A considerable epidemic of dengue prevailed in Brazil in the summer of 1846, and to a less extent in each of the following three years, likewise at the same season. It also appeared in Peru in 1852—here, too, as a precursor of yellow fever.

The disease found another remote field in Lower Egypt and on the coasts of Arabia, where, subsequently to the epidemic of 1779 which we have mentioned, it was first observed at Cairo, in 1799, by Gaberta, and then occurred again, after a long interval, in 1835 and 1845, under the name of "knee-evil" (Pruner).

Next to these we see India most frequently visited by epidemic dengue; first, in the year 1824, it appeared during the hot season in the southern parts; in the rainy season in Calcutta, and spread thence along the Ganges, over the largest portion of the Bengal and Madras Presidencies. In the succeeding year, and again in 1836, the dengue returned to Calcutta during the rainy season, and again spread as an epidemic in the same locality and in the Bengal Presidency. Epidemics were next recognized in the years 1853 and 1854.

In 1860 the dengue appeared at Martinique, first among the ships which had recently arrived and in the garrison. According to Ballott, one hundred and twelve cases of the disease occurred among the four hundred men constituting the garrison. The disease did not extend into the city until later.

In Spain, too, the disease was recognized in the years 1864–1868 under the name of La piadosa (the exciter of compassion) or La pantomima. It appeared first at Teneriffe, at Cadiz, and at Seville, and in the following years in Andalusia and some other provinces of Spain.

The last outbreak of dengue occurred very recently, in the years 1871 and 1872. The epidemic first appeared in Arabia, where it was called Aburuka-Bah (Father of the knee), and was observed in 1871 by Read, especially in Mecca, Medina, and Aden. In Aden it prevailed epidemically for over seven months; of the garrison of nine hundred men, seven hundred had the disease. Sporadic cases showed themselves for a still longer time. As a result of the constant communication with ports in the southern seas, the disease was carried in 1871 to Zanzibar and other points on the African coast (Read, Christie, and others). Vauvray communicates the intelligence that it prevailed at the same time at Port Said, *where, however, it was epidemic every year at the season of the date-harvest*, and thus acquired the name of date fever. In November, 1861, it reached Java, and became localized for several months, especially in Fort Willem I. and its vicinity.

Furthermore, it was imported directly into Bombay and Cananore by two troopships (M. Sheriff). Thence the epidemic extended in 1872 through all India, though confined at first to the Madras and Bombay Presidencies, especially along the course of the railroads. Even in other English stations in Burma, China, and Nepaul cases of this highly remarkable epidemic were found. In its diffusion the disease was as wide-spread as it was intense. In some of the cities attacked, scarcely an inhabitant remained exempt. It prevailed most violently and most extensively in Madras, where not a house escaped, and it attacked equally both sexes and all ages (the oldest patient was eighty years, the youngest two months old), and persons of every condition, even up to the highest classes. The fever attained its greatest extent in Madras in September and October. According to Mooden Sheriff there came into the public dispensaries for treatment in July, twenty-five cases; in August fifty-two; in September, one hundred and seventy; in October, one hundred and fifty-seven. After a heavy fall of rain, between the 13th and 14th of October, the disease suddenly abated to such a degree that only slight sporadic cases occurred up to the end of the year; only four in November and December.

The *etiology*, in regard to the exciting causes, appears completely obscure. The question whether the dengue is contagious in the ordinary sense still eludes decision, although the disease is capable of being conveyed to a distance by articles which are constantly passing from hand to hand, as was made evident by the mode of diffusion of the last epidemic especially. Certain observers, as M. Sheriff, favor the assumption of its contagiousness, without, however, being able to adduce positive proofs; others, as Thaly, deny it decidedly. Most physicians who have had an opportunity of observing the disease refrain from expressing an opinion about it.

In certain cases the affection appears as the precursor or companion of yellow-fever epidemics in the maritime countries attacked by this disease (Avé-Lallemand), but it has also occurred, independently of the latter, there, as well as in mountainous regions otherwise healthy. Moreover, no uniformity can be assigned in regard to the seasons of the year especially favored by dengue, only it appears hitherto to have been essentially limited to tropical regions or those lying near the tropics. Finally, all races and nationalities, with the exception of the negro (de Wilde), appear to be equally subject to the affection (M. Sheriff counted among the patients in the public dispensaries fifty-seven

Europeans, two hundred and thirty-five Mohammedans, and one hundred and twenty Hindoos). In the last epidemic persons recently arrived appeared to be somewhat more lightly attacked by the disease than residents already acclimated.

Symptomatology.

The features of the disease as given by the first observers have been modified and enlarged by more recent observations, but still exhibit many defects.

The disease begins after a stage of incubation of three to five days (M. Sheriff), while, as estimated, in about half the cases it begins suddenly. In the same way it often has a prodromal stage of one to three days' duration, with slight chills, headache, pains in the back and along the spine, and a burning sensation in the stomach.

With the beginning of the fever there follow the painful swellings of the joints peculiar to the disease, together with turgescence of the skin, the face, and the neck; less frequently an exanthem now shows itself, the temperature advances quickly and significantly, and reaches its height within the first twelve to twenty-four hours. A bodily heat of 106.7° F. and 107.6° F. is not seldom observed in the axilla (Müller and Manson and others); once even, according to an anonymous French naval surgeon, a heat of 108.8° F. The fever is now continuous, the pulse exceedingly frequent (120 to 140) and very strong, the breathing quickened (28 to 30), and the skin dry and of a burning heat.

After two to three days, sometimes in less time, but sometimes only after five to seven days, the fever generally abates suddenly, often with the occurrence of critical symptoms, as in the case of relapsing fever, such as profuse sweats, or diarrhœa, or epistaxis (Müller and Manson and others). The temperature sinks, but still remains somewhat above the normal, while the skin becomes moist, and the other symptoms of the disease so far subside that the patients do not hesitate to leave the bed, and even the chamber.

This favorable state, which seems to be analogous to the stage of intermission in relapsing fever, is sometimes wanting (Ballot).

According to de Wilde, it generally lasts one or two days. Now the second paroxysm follows, with a new and rather moderate elevation of temperature of a remitting character, and simultaneously with it an exanthem shows itself upon the skin. Less frequently there is a relapse of the joint-affection. The symptoms last for some two or three days. The disease, as a rule, comes to an end with a gradual subsidence of the symptoms, the second paroxysm seldom extending over a longer time.

In exceptional cases the first paroxysm is accompanied by a peculiar *affection of the joints and limbs.* In severe cases it involves all the joints ; often, the pains in the bones and muscles are described as "boring" and "breaking" with more or less stiffness of the affected limbs and swelling of certain tendons.

The affection attacks large and small joints without distinction, seldom fewer than six to eight at the same time. In succession the joints of the hand, foot, and knee are first attacked, the spine and fingers, then the toes and the joints of the elbow and shoulder. It happens most frequently that the hand cannot be closed, and it is particularly difficult to bend the fingers in the morning. The joints affected are much swollen, red, immovable, painful, and highly sensitive on pressure.

Most authors characterize the pains as diffuse, rheumatic, or rheumatoid ; still de Wilde several times observed the occurrence of single painful points, or where nervous trunks, as, *e.g.*, the ulnar, were specially affected ; and Thaly also regards them as gouty. It is very noticeable with what celerity the pains change and diffuse themselves. According to Dunkley, while the patient in the morning complains only of severe headache and pains in the hands, by evening the joints of the foot or the knee are chiefly affected. According to Hirsch, in three autopsies serous infiltration of the connective tissue in the neighborhood of certain joints was twice found, and redness of the crucial ligaments of the knee once.

This affection diminishes with the subsidence of the fever; sometimes, however, it is persistent, and yields only after weeks, or even months.

In many cases, conformably to the two distinct paroxysms, a *primary* and *a secondary exanthem* are to be distinguished, according to the time of their occurrence (de Wilde and others). The primary, less often observed, appears and disappears simultaneously with the fever; but the eruption on the skin quite regularly accompanies the second paroxysm. It is accompanied by decided itching of the skin, and is variable in form; sometimes maculated, like roseola; sometimes elevated, like lichen tropicus; evenly distributed, resembling measles or scarlatina, or in blotches, reminding one of urticaria. The bright red color of the affected parts often passes into crimson on the palm of the hand and sole of the foot (M. Sheriff, de Wilde, and others).

The affection of the skin is of variable extent, limiting itself sometimes to single spots, or again extending over the greater portion of the body. Most frequently it is localized upon the face (where in the neighborhood of the eyes it induces marked conjunctivitis and lachrymation), upon the neck, chest, and arms; also upon the feet and hands. It lasts only a few hours, or two to three days, and generally leaves behind a furfuraceous desquamation, which may continue for a long time on the tips of the fingers in certain patients (de Wilde).

According to Christie's description, in severe cases we also frequently find the *mucous membrane* of the nose, mouth, and throat inflamed; and the *lymphatic glands* in the neighborhood of these, and of the skin affection, are often the seat of transient swelling.

Of the remaining symptoms of the disease, the loss of *appetite* which accompanies the whole course of it, and the great increase of *thirst*, assume prominence. The patients complain of a disordered taste and of nausea; it seldom amounts to vomiting. The tongue is coated white or of a greenish yellow (according to Dunkley silver-white). *Constipation* appears generally to last through the first part of the fever, but to subside after the crisis, which is sometimes marked by diarrhœa.

During the fever the *urine* shows a high specific gravity, a dark color, but no albumen; from being previously scanty it often increases materially in quantity at the time of the crisis (Müller and Manson).

During the fever the patients are restless and sleepless, and complain of pains in the head, which, according to the descriptions, especially involve the forehead. Certain ones are delirious at night. Severe cerebral symptoms are not observed.

A case of M. Sheriff's, ending in recovery, is worthy of mention, as it was marked by spasms of the respiratory muscles; they recurred at intervals of two or three minutes, and lasted for a time, whereby the patient was highly distressed.

Next to the *emaciation* which quickly appears, the patients experience a marked sense of *loss of strength*, especially in the legs, which is often perceived early, and is even long complained of in convalescence.

Not seldom there remains after the fever *an affection of the heart*, which has been repeatedly considered to be pericarditis, even by M. Sheriff and Dunkley, but it never leads to a fatal issue, and subsequently disappears.

Beyond two or three cases of pleurisy observed by Dunkley, the *respiratory organs* have been found completely intact.

Certain cases of *pregnancy* at various stages resulted in abortion.

In children the features of the disease are often somewhat nodified by the shorter duration of the fever, but especially by epeated or persistent *convulsions*, which may even lead to eath.

In adults the *prognosis* is very favorable; fatal cases seem to ave been observed only very exceptionally. Out of 165 patients f de Wilde's none died, while among the children affected, some ases ended fatally from convulsions (de Wilde, M. Sheriff, and thers).

The *course* of the disease may be generally divided, according o the more recent communications, into periods of the first ebrile access (two to three days), the intermission (one, two, or hree days), which, however, is not seldom wanting, and the econd febrile stage (two to three days), whereupon convalescence egins. This latter, however, is now and then disturbed for a ong time by great weakness, emaciation, and persistent affecions of the joints.

Veritable *relapses*, after an interval of two or three weeks, ere repeatedly seen to occur by Sheriff; they seem to run a ilder course than the primary affection.

In the first paroxysm the disease sometimes appears to offer certain resemblances to articular rheumatism ; in the second, to scarlatina. Still, the diagnosis can scarcely be difficult.

Scarcely any conjectures have been raised as to the *nature* of this obscure affection. Only there remains, when it follows a typical course, a certain striking likeness to relapsing fever, and this certainly calls for an examination of the blood.

TREATMENT.

Among therapeutic procedures it is worth noting that, according to M. Sheriff, quinine (gr. iij.-v. every three or four hours) seems to have some influence in shortening the disease ; taken as a prophylactic it is said to furnish great protection against it.

The treatment is generally begun, according to the practice in the tropics, with an emetic or purgative ; then quinine, diaphoretics, and warm baths are employed (M. Sheriff), or laxatives —rhubarb, aloes, sulphate of magnesia, and the like—(de Wilde); while the French naval surgeon already mentioned, Christie, and others recommend belladonna in large doses as very efficacious against the pains in the joints ; also strychnine, phosphoric acid, and colchicum. Cold baths also are in repute. After the disappearance of the fever, Christie and others give iodide of potassium. For the relief of the pains in the limbs, friction with spirituous and stimulating liniments is employed, and chloroform, electricity, tonics, etc., to do away with the great weakness which often lasts for a long time.

To prevent the spread of the disease, M. Sheriff advises isolation of the patients and quarantine of the districts affected.

INFLUENZA.

EPIDEMIC CATARRHAL FEVER, LA GRIPPE, GRIPPE.

iterature of influenza, which formerly excited a high degree of general inter-
st, has reached such an extent that we must renounce the idea of presenting it
detail in this place. It is principally comprised in the following mono-
raphs: *Saillant*, Tableau historique et raisonné des épidémies catarrhales, vul-
airement grippe, depuis 1510 jusque et y comprise celle de 1780. (Detailed reci-
l of the older literature.)—*Zeviani*, Opusculo sul catarrho epidemico. (Memor.
i Mathem. e di Fisica della Soc. Ital. delle Scienz. T. XI. Modena, 1804.—*H.*
chweich, Die Influenza. Berlin, 1836.—*G. Gluge*, Die Influenza oder Grippe, nach
en Quellen histor.-pathol. dargestellt. Minden, 1837. (Good critical review.)
-*A. Hirsch*, Handb. der histor.-geograph. Pathologie. I. 277–300. Erlangen,
860. (Especially treating of the epidemic relations.)—*J. Fuster*, Monographie
linique de l'affection catarrhale. Montpell., 1861.—Next to these, prominence is
be given to the following works: *Cullen*, Synopsis Nosologiæ Method. Ed.
uinta, 8vo. Edin., 1792.—*Ph. L. Wittwer*, Ueber den jüngsten epid. Catarrh.
ürnberg, 1782.—*N. Webster*, Hist. of Epid. and Pestilential Diseases. Hartford,
799. Vol. II.—*Adams*, An Inquiry into the Laws of Epidemics. Lond., 1809.—
tit, In Diction. des Scienc. Méd. T. XIX. p. 351. Par., 1817.—*Most*, Influenza
uropæa oder die grösste Krankheitsepidemie der neueren Zeit. Hamb., 1820.
-*Foderé*, Leçons sur les épidémies et l'hygiène publique. Par., 1822.—
chnurrer, Chronik der Seuchen. Tübingen, 1823.—*J. Frank*, Prax. Med. uni-
ers. præcept. Vol. I. Sect. II. p. 50 et seq. Lips., 1826.—*Sprengel*, Versuch einer
ragmat. Geschichte d. Arzneik. T. V. 3 Aufl. Halle, 1828.—*Naumann*, Handb.
med. Klinik. I. Bd. p. 424 et seq. Reutlingen, 1832.—*Zlatorovich*, Geschichte
es epid. Catarrhs (Influenza), welcher im Frühjahr 1833 in Wien grassirte, und
ber sein Verhältniss zum stationären Genius der Krankheiten. Wien, 1834.—
zanam, Hist. Méd., etc. 2d ed. T. I. p. 29–218. Par., 1835.—*Richelot*, Recherch.
ur les épidém. de grippe, etc. Arch. gén. T. 37 and 38. 1835.—*Dunglison*, Med.
eview. T. XX. 1 Ser. p. 444.—*Bouvier*, Annal. d'hygiène publ., Avril 1837.
. XLIII.—*Nonat*, Recherch. sur la grippe et sur les pneumonies observées pen-
ant le mois Février 1837. Par., 1837-8.—*Lereboullet*, Rapport sur l'épidém. de
rippe, qui a régné à Strassb. pendant les mois de Janv., Févr. et Mars 1837.
aris et Strassb., 1838.—*Copland*, Dict. of Pract. Med. New York, 1846. Vol.
I.—*Graves*, Clin. Lect. on Pract. of Med. 2d ed. Vol. I. Dublin, 1848.—*Can-*
tatt, Handb. d. med. Klin. II. Bd. 2. Abtheil. Erlangen, 1847.—*Toulmouche*,

De la grippe épidémique, qui a régné en 1837, etc. Gaz. méd. de Par., 1847, p. 858.—*Theoph. Thompson*, Annals of Influenza; prepared for the Sydenham Soc. Lond., 1852.—*Forget*, Gaz. méd. de Strassbourg. 1858.—*H. Häser*, Historisch-patholog. Untersuch. I. 58.—*Legrand*, Sur la grippe; constitution méd. etc. Par., 1860.—*H. van Holsbeck*, La grippe et son traitement. Annal. de la Soc. de Méd. d'Anvers. Janv. 1861.—*Biermer*, In Virchow's Handb. der spec. Path. u. Ther. Erlangen, 1865. V. 1st Part, 592. (Very comprehensive statement.)—*Corradi*, L'Influenza ovvero Febr. cat. epidem. d'ell' an. 1580 in Italia, con nuovo docum. illustr. Milano, 1866.—*Hjaltelin*, Edin. Med. J., May, 1866.—*Hall*, Lond. Epidemiol. Transact., II., Part I., p. 69. 1866.—*Gérard*, De la Contag. de l'influenze. Ann. 575. 1866.—*Tigri*, Annal. Univ. Vol. CCII. p. 677. 1867.—*Montard-Martin*, Gaz. d. hôp. No. 26, p. 101; No. 29, p. 113. 1867. —*Petit*, Quelques mots sur la grippe. Ibid., No. 37, p. 147.—*Vincent*, De différ. formes de la grippe, 34, p. 4. Paris, 1868.—*Lydtin*, Infl. der Pferde. Fuchs M. S. 179. 1869.—*Richardson*, St. Andrew's Med. Grad. Assoc. Trans., II., 1869, p. 234.—*C. Handfield Jones*, Brit. Med. J., July 23, p. 81. 1870.—*J. O. Webster*, Report of an Epid. of Infl. Bost. Med. and Surg. J., June 8, p. 377. 1871.—*H. Gintrac*, In Nouv. dict. de Méd. et de Chir. pratiques. T. XVI. p. 728. Par., 1872.

INFLUENZA is an epidemic disease which is essentially characterized by catarrh of the respiratory, and generally also of the digestive organs, by great and rapidly developed weakness, pains in the limbs, severe headache, as well as by serious nervous symptoms, and fever of greater or less intensity.

The disease is principally distinguished from simple epidemic catarrh by its universal diffusion: it spreads with the greatest rapidity over extensive districts of country, and even over whole quarters of the earth, and in those regions in which it occurs it spares only a small fraction of the population. It appears unconnected with atmospheric influences, especially with those which otherwise are wont to occasion catarrhs. Moreover, the course of the disease is marked by important lesions of function, which would not correspond to a simple respiratory catarrh. The affection, therefore, in spite of this special localization, has been regarded by numerous authors as a general febrile disease, which is dependent on miasmatic influences as yet unknown to us.

Its names, which have a pre-eminently popular origin, are very numerous; Gluge and Schweich especially give a comprehensive collection of them. Many of the older appellations are significant; such as Modefieber (fashionable fever), Schafshusten (sheep-cough), Hühnerziep (crowing), Blitzkatarrh (lightning catarrh), or Barraquette, Petit-Courrier (the little courier), la Follette (the frolicksome), la Générale. (Coqueluche is used ambiguously.) Other names signify the region where the disease was thought to originate: Spanish catarrh, Russian catarrh, etc.

The name now in common use in Europe, "La Grippe," comes, according to Biermer, from *agripper* (to seize), according to Joseph Frank, from the Polish word *chrypka* (raucedo). The word "Influenza" is of Italian origin; perhaps it refers to the assumed influence of the climate or atmosphere, or from the further signification of the word (something fluid, transient, or fashionable), to the name commonly used in the epidemic of 1709.[1] According to Biermer, Pringle[2] first designated the disease by this name.

HISTORY.

The history of influenza can be traced back with certainty nly to the beginning of the sixteenth century. Since that time nly have the records been particular and distinctive enough to nable us to infer its existence unequivocally from them. Yet it eserves to be brought into prominence, that even as early as the inth century not a few epidemics of catarrhal fever, Italian ever, and the like are cited, which possibly refer to the same isease. In the writings of classical antiquity no statements can e established as referring to it.

With the year 1510 begins a series of epidemics, the wide distribution of which has been reached by no other acute infectious disease. In this respect it forms a diametrical contrast to the ummer-rash which has been spoken of before. Up to the year 870 more than ninety epidemics, connected to a greater or less extent, have been described, which generally extended over whole countries, frequently over several quarters of the earth. They returned at indefinite periods, and affected every season nd latitude, although the colder climates somewhat more frequently. As a rule, they advance in a great wave, in our hemiphere passing from the east and north-east towards the west. More rarely they seem to have taken another direction or to have radiated from particular centres. Sometimes they occur simultaneously at several points of the earth.

---

[1] *Slevogt*, Prolusio, qua die Galantriekrankheit oder das Modefieber delineatur. Jen. 1712.

[2] Observ. on Diseases of the Army. Lond., 1752.

In rare instances the epidemics are preceded by the appearance of sporadic cases. Much more commonly they simultaneously attack with great rapidity a large number of the inhabitants of the affected districts, so that in populous cities business is often suddenly destroyed as by a shock, and the number of the sick is soon counted by thousands. They are only very exceptionally limited, as in the epidemic of 1836–37 in St. Petersburg and Paris, to the environs of certain cities, or, as in certain smaller epidemics, to particular classes in the population (*e.g.*, in the epidemic of July, 1833, in Novara, according to Galli,[1] it spread from among the population to the garrison, and in 1803 was confined to the civil inhabitants, while it rather avoided the military).

This peculiarity of influenza is so striking that many authors have been led to the opinion that the numerous epidemics, limited to more narrow precincts, of which Hirsch (loc. cit., p. 286) enumerates nearly eighty, were not at all to be regarded as of this disease, although when looked at apart from its universal spread they actually differed from it in no respect.

The epidemics lasted, as a rule, four to six weeks; less often they ran their course in a shorter time, and only exceptionally, as in Paris, in 1831, did they last, according to Fuster, for nine to ten months. They rapidly reached their height, and their ending was almost equally as sudden as their beginning; yet sometimes they returned to a district which had already been once attacked.

Among the most important epidemics which have been carefully described are the following:

The epidemic of the year 1510 spread from Malta in a direction from south-east to north-west over the whole of Europe, and attacked everybody. Yet, with the exception of children, very few died; among them Anna, consort of Philip I. The life of Pope Gregory XIII. was also endangered. The disease was accompanied by severe pains in the supraorbital region, by delirium, gastrodynia, syncope, subsultus tendinum, and a black coat on the tongue (on the seventh to eleventh day). On the abatement of the disease diarrhœa and sweats usually set in. Bleeding and urgatives proved injurious, therefore blisters were generally used. Two each to he arms and legs, and one to the occiput.[1]

---

[1] Schmidt's Jahrb. VI.

The epidemic of the year 1557 spread from Asia across Europe, and across the cean even to America. It broke out late in the autumn, and lasted during the winter, spring, and summer. Almost everybody was attacked (in Nismes) on the ame day.[2] Its symptoms were more or less severe fever, headache, catarrh, sore hroat, cough, and great weakness. If, after bloodletting and the use of expec-orants, an offensive sweat occurred, the patient recovered; but if the fever continued with great exhaustion, he succumbed to the disease.[3] The disease was often terminated by a diarrhœa.[4] The affection was most destructive in Sicily.

The epidemic of the year 1580 spread from south-east to north-west over Asia, Africa, and Europe. The disease came from Constantinople and Venice to Hungary and Germany, thence to Norway, Sweden, Denmark, and Russia. In Spain it prevailed during the whole summer; in Italy from August to the end of September. The issue was almost always favorable, and was marked by copious sweats. In many places, on the contrary, the disease ran a very severe course; in Rome, *e.g.*, it claimed about 9,000 victims, and Madrid must have been almost depopulated by it. Wier[5] ascribes the great mortality in Rome to the venesections; "the best mode of treatment consisted in trusting entirely to nature." The number of patients was very great; in many regions, as in Saxony, nearly four-fifths of the population were attacked.[6]

In the year 1591 the influenza appeared anew in Germany; in 1593 in Holland, France, and Italy; in 1626–27 in Italy and France; in 1642–3 in Holland; in 1647 in Spain and North and South America; in 1655 again in North America; in 1658 and 1675 in Germany, Austria, England, etc.; in 1688 in Great Britain; in 1693 in Great Britain, France, and Holland; in 1709 in the countries of Central Europe; in 1712 it was very prevalent in Denmark, Germany, and Italy.

The epidemic of the years 1729–30 was one of the most wide-spread in Europe; in five months it overran Russia, Poland, Hungary, Germany (in Vienna more than 60,000 persons were attacked by it), Sweden, and Denmark; in the autumn it reached England, France, and Switzerland, and from there Northern Italy; it visited Rome and Naples in February, and is said to have made its way through Spain even to Mexico. Its essential manifestations were, pains in the limbs, catarrh, oppression of the chest, hoarseness, and cough; sometimes there also occurred cerebral symptoms, delirium, somnolence, attacks of faintness, etc. In some cases a petechial eruption was observed about the fourth to the seventh day (was spotted fever prevailing at the same time?). The disease was often characterized by turbid urine, copious sweats, bilious stools, hemorrhages from the nose, etc. In Switzerland children and aged persons were almost the only ones that died.[1]

---

[1] *Sennert*, De abd. rer. caus., conf. *Ozanam* and others.

[2] *Mercatus*, De Corp. hum. Affect. II. 1. Op. Venet., 1611.

[3] *Riverius*. Op. Ludg. Batav. 1663. Appendix.

[4] *Valleriola*, Loc. Med. Com. App. II.

[5] Observationes. Amstelod., 1660.

[6] Reports on the subject in *Salius Diversus*, Opusc. Med., *Riverius* and others; conf. *Gluge*.

The epidemic of the years 1732–33 spread from Saxony and Poland, in the middle of November, over Germany, Switzerland, and Holland, and reached Great Britain in December. Towards the end of January it advanced in one direction south-easterly (to Paris, Italy, Naples, Madrid), and in the other across the ocean to North America; then extended southwards to Jamaica, Barbadoes, Mexico, and Peru. The epidemic also continued during the years 1834–37, overrunning north-eastern Europe and spreading towards the south-east. In Italy the brain symptoms predominated—headache, mental disturbances, perversions of the senses of smell and taste, aphonia, etc. Its course was generally favorable; the disease came to an end on the third to the fourteenth day, with sweating, epistaxis, abundant expectoration, or defluxion from the nose. The aged, however, the asthmatic, and the phthisical succumbed almost everywhere. In Scotland the "three forms of the disease" (encephalica, thoracica, and abdominalis) were observed; in England more frequently the encephalic form, with catarrh, sneezing, sleeplessness, dizziness, headache, slight delirium, ringing in the ears. Almost all observers ascribe the epidemic of 1732–37 to the continual changes in the temperature.[2]

In the years 1737–8 the influenza again prevailed in England, North America, the West Indies, and France; in 1742 in Germany; in 1742–3 in Switzerland, Italy, France, Holland, and England. In 1757 it showed itself generally in North America, and appeared in the West Indies, France, etc. In 1758 it attacked France and Scotland; in 1761 it prevailed widely in North America and the West Indies.

The epidemic of the year 1762 extended very generally through Germany (scarcely a tenth of the population remained free from it), through Holland, France, Italy, and Great Britain. In 1767 the disease again appeared in Germany, France, Italy, England, Spain, and America; in 1772 in North America.

The epidemic of the year 1775[3] again overspread Europe. In Germany the "abdominal" form prevailed. From England the disease came to America. In 1778–80 we find the influenza in France, Germany, and St. Petersburg; in 1781 in Wilna, North America, and China (?).

That of the year 1782 belongs among the most remarkable of the epidemics of influenza. It began in Russia; according to others it must have come from America (Webster), or from Asia into Russia. On January 2d, the thermometer rose in the course of the night from 35° below zero to 5° above zero, and on the same day about 40,000 persons fell sick with influenza, the explanation of which, it was believed, was to be sought in the sudden change of temperature. According to J. Frank, the epidemic must have shown itself in St. Petersburg in February, and have come there from Astrachan across Tobolsk. From here it spread over Sweden,

---

[1] *Fr. Hoffmann, Loew;* Ephemer. cur. nat. and others; conf. *Schnurrer.*

[2] Conf. the literature in *Gluge, Hirsch,* and others.

[3] *Stoll,* Rat. med. Vienn., 1777. *Saillant* and others.

Germany, Holland, and France; in the autumn it was in Italy, Spain, and Portugal, and did not even spare the English and Dutch ships upon the high sea. In Vienna the disease attacked three-fourths of the population, and occurred so suddenly that it was called on this account "Lightning Catarrh." Its course was marked by an extraordinary enfeeblement and great pain in the back, throat, sternum, and larynx. Children remained almost exempt. There were often relapses, pneumonias, and inflammations of the bowels.[1]

To continue: the years 1788–90 were marked by numerous epidemics in Europe and America, some of which returned, and the years 1798–1803 by a new outbreak of the disease, which began in the north-eastern part of Russia, and spread over the whole of that country, Germany, England and France, and a part of Italy. From the year 1805 to the year 1827 we again meet with a great series of epidemics in Europe and America; only a few years were free from them.

The epidemics beginning in 1830 are distinguished for their universal diffusion and their rapid succession. The extensive literature on the subject points to the general interest which they excited. The reports state that the first outbreak of the disease in 1830 occurred in China; in September it appeared in the Indian Archipelago (Manilla), and in November and December in Russia (Moscow); in January, 1831, upon the Great Sunda Islands, and at the same time in St. Petersburg; in February in Curland and Livonia; in March in Warsaw and the north of Java. Further than this, in April the disease affected East Prussia and Silesia; in May Denmark, Finland, and a great part of Germany, also Paris; in the following month also a great part of England, Sweden, and at the same time Farther India (Singapore) and the Indian Archipelago. In July the influenza raged in Würtemberg, in Switzerland, in Toulouse, England, Sweden, and Farther India (Penang); and in the beginning of winter in Italy and in certain regions of North America, where also it was still observed in January and February, 1832. After it had become extinct in Europe, it still prevailed in Hindostan, and occurred anew in Russia in January, 1833; in February it was in Galicia and East Prussia (Memel); in March in Egypt and Syria, in Prussia, Bohemia, and Warsaw; in April in many districts of Germany and Austria, also in Pesth, Copenhagen, Jutland, France, and Great Britain. Up to July the catarrhal fever was still diffused in Germany and Upper Italy; and in September in Switzerland and France (Department of the Moselle), and in November in Naples.

In the year 1836 the influenza showed itself in December in Russia, Sweden, and Denmark; in January, 1837, in London, and in a very brief time spread over all England, Germany, and France. In January the disease appeared in Berlin, somewhat later in Dresden, Munich, and Vienna. At the beginning of February Switzerland and France were attacked; at the end of March Spain also (Madrid). In London almost the whole population was attacked, and the mortality was very great. This epidemic also spread to the southern hemisphere, and prevailed at Sydney and the Cape of Good Hope at the same time that it visited the north of Europe; at directly the opposite season of the year therefore. In the severe cases, there occurred pains in the loins and joints, and very great loss of strength; the catarrh was suffocative, and many patients died asphyxiated. Most observers were astonished at the great mortality of this epidemic.[1]

---

[1] Conf. *Strack*, Diss. de cat. epid. a. 1782. *Moyant*, 1782. *Grant*, Observat., etc., Lond., 1782. *Falconer*, An Account, etc., Lond., 1782, and others, especially *Wittwer*.

From that time up to 1850–51 we find more or less extensive epidemics almost every year, with few exceptions (Hirsch). Also in 1857–58–60; in 1864 in Switzerland (Biermer); in the spring of 1867 in Paris (M. Martin); and since then separate outbreaks in North America have been described by Webster and others. As for the rest, the more recent epidemics have run a less dangerous course, and therefore, like the last epidemic of 1874 in Berlin, have been scarcely mentioned in literature.

ETIOLOGY.

Extensively as literature has been occupied with the histories of epidemics of influenza, the gain in well-established facts as to its etiology is still exceedingly small.

In regard to *predisposition*, everybody is agreed in stating that the epidemics attack the population without distinction of age, sex, constitution, or condition, no matter whether their occupation be more in the open air or within doors. Only in one direction a great portion of the observers have established a certain rule. In places where the disease occurs, it is said to attack first the female population, next, the adult males, and lastly, the children. In some epidemics the children, moreover, remain strangely exempt. Weakly and nervous individuals are said to be more quickly and more often attacked than the robust, and, according to the views of some, aged, infirm persons are particularly liable to attack. The latter view is perhaps based on this, as Biermer supposes, that from the great danger of influenza in old age we pay greater attention to it. Neither do those persons remain exempt who are suffering from the most various acute and chronic diseases, internal or external. In fact, those dwelling in every latitude and belonging to every race are equally subject to the influenza.

In opposition to this there is only to be adduced a statement of Graves, viz., that fluenza does not appear during the febrile stage of continued fever, but seizes the tients at the beginning of the period of convalescence. According to some obser- tions made in Iceland[1] and in the Antilles (according to Barclay), the natives ly appear to have been attacked, while strangers remained free.

---

[1] Conf. *Landouzy*, Mém. sur l'épid. de 1837, *Rebouillet*, *Heine*, *Günther*, *Piorry*, and various others in *Canstatt*, *Hirsch*, *Fuster*, etc.

No connection with *atmospheric conditions* has been estab- shed, although some observers declare particularly violent hanges in temperature, or foggy weather and the like, to have een prevalent at the time of epidemics; other observations show he direct opposite of this. Herein lies an essential difference rom non-epidemic catarrh, since this must be referred to 'catching cold," and therefore is pre-eminently traceable to amp, raw air. Influenza, on the other hand, prevails in hot and ry seasons, as well as in countries where, in virtue of the high ean temperature and the slight degree of dampness, catarrhs re exceedingly rare; *e. g.*, in Egypt and other tropical regions. ne fact, however, is established in many ways, and is in accord ith the observations as to the spread of many epidemics from ast to west, viz., that east and north-east winds have often pre- ailed at the time of the influenza.

At all events, the disease is unconnected with any *local cir- umstances* whatever of elevation, condition of the soil, and the ike; without assuming this, its wide distribution would be nexplicable.

Little is known of the *exciting causes* of influenza.

The assumption of *contagiousness* has been abandoned just s often as it has been established; the occurrence of cases in roups, the fact that often all, or at least the majority of the nhabitants of a house, of a street, or even of a quarter of the ity are attacked simultaneously, or at uncertain intervals unning through days or weeks, these may have turned our houghts in that direction. This observation also might be dduced in proof of it, that many times in secluded places, lthough a considerable number of persons lived there together, *g.*, in cloisters, prisons, and the like, no cases of sickness ccurred, so that it appeared that possibly even the seclusion pro- ured immunity. Other instances point in the opposite direction—many of which were deduced especially from the epidemic beginning in 1830—as that even complete seclusion from intercourse, or actual shutting up in the dwelling, furnished no security against an attack. On the other hand, even such persons as came into immediate contact with the sick often remained wholly unaffected, such as surgeons and nurses, and even bed-fellows and room-mates. The spread of the disease, too, is generally much too rapid to be at all explicable by contagion (Biermer).

---

[1] According to *Schleisner*, in his Medical Topography of Iceland. Copenhagen, 1849. chmidt's Jahrb., 70.

Moreover, it is especially worthy of note that the propagation of influenza is not determined by human intercourse. Even the most crowded highways and places have often been exempt or only slightly affected by the catarrh, and dwelling-houses, barracks, factories, and the like, situated at the very gates of cities, remained completely untouched, sometimes in great epidemics and in spite of uninterrupted communication.

In their progress the epidemics of influenza show, as alleged, a great diversity. Some authors estimate that it moves forwards as fast "as a rider;" at different times it travels exceedingly slowly, or with the greatest rapidity; often it even attacks districts far removed from one another with inexplicable leaps, or appears simultaneously at different points of the earth's surface.

With such want of agreement in the observations, no etiological theory whatever has been developed upon a satisfactory basis. The causes of influenza seem completely *sui generis*, and are as unknown to-day as they were centuries ago. The great number of hypotheses is the best proof of this.

The causation of the epidemics has most frequently been sought in atmospheric agencies; it has always and with good reason been believed that so general a disease could be disseminated only by means of the most general medium, the air.

According to Crato and Mercurialis (compare Gluge) the air at the time of the epidemics is "putrefied;" others speak of a "contagium" or "miasm" in the air, of caustic, saline, sulphurous, or nitrous contaminations of it, which were referred in part to volcanic influences and also to fog, vapor, and the like, to phlogisticated air, to increased or diminished electricity, terrestrial magnetism, and other causes. The latter assumption especially was widely diffused by Wittwer, Schweich, and thers. Schönbein, Spengler, and others referred the epidemics to the altered conition of the air as regards ozone, a view which met with a decided refutation from chiefferdecker, of Königsberg, as the result of sufficient observations. An animated contagious material also has often been spoken of. In the epidemic of 1782 the lea was spread abroad that the air was infected by an insect, called therefore Grippe," a view which Grant refuted in a special paper.[1] Elsewhere, as in Vienna, where the Medical Council distinctly expressed itself to this effect, the disease was referred to an insect said to be swallowed with the water; and the philosopher Kant imagines that we may believe with much probability that the Russian trade with China had imported some kinds of injurious insects which gave rise to the nfluenza.

The most important question, at all events, in the etiology is, whether in the different great marches of the epidemic the individual outbreaks are connected, are, in fact, the operation of a common cause, or run their course independently of one another, i.e., whether, as Biermer is inclined to consider, the single local epidemics are to be referred to an indigenous origin. It seems n accordance with this idea that, while the disease spreads rapidly in its local diffusion, it advances comparatively slowly on a grand scale over countries and seas without being essentially influenced by human intercourse or the direction of the wind. In other cases again, it remains limited to narrow circuits.

Again, these observations may easily have a different significance. Although indefinite intervals often exist between the single local epidemics, their immediate connection is still highly probable in the great majority of cases, although often difficult of demonstration. The case is just the same as it was with cholera before we began to pay attention to its etiological relations; and in the case of an affection which is, on the whole, nsignificant, like influenza, no urgent impulse has bidden us undertake it. Influenza, too, like cholera, does not occasion any independent endemics. The catarrh now and then occurring in separate places (as, *e.g.*, in Iceland and Faroe), besides being wanting in epidemic diffusion, presents no serious symptoms. Much more does the complete correspondence of symptoms render the association of single epidemics within a definite period highly probable, the more so as they often move forward in a definite direction (as a general thing from east to west).

---

[1] Observ. on the late Influenza, etc. Lond., 1782.

Many of the earlier authors arrived in this way at the idea of a specific principle of disease, which is developed from time to time somewhere in high northern or eastern regions, and thence travels abroad. Besides, Biermer calls attention to this, that the apparent contradictions of rapid diffusion and slow migration, of general extension and local limitation in the history of influenza would be easy of explanation on the theory of a *living miasm*, which would be capable of being carried onward by the air, but had an independent existence of its own, and would find in certain places conditions more favorable for its development than in others. An established basis of facts is meanwhile wanting for this hypothesis, as well as for all the others.

Certain accounts are found in the literature of the disease, according to which other epidemic or endemic diseases have vanished on the appearance of influenza. Thus, as Smart[1] relates, an epidemic of scarlatina became extinct during the influenza, but afterwards returned. Small-pox[2] also, and spotted fever[3] have declined on the appearance of influenza. Gallicio[4] and Panum made the same observations regarding intermittent fever in the epidemics of 1831 and 1833. On the other hand, Escherich, Stosch, van Dembusch, and Galli assert the opposite, that the influenza not seldom degenerated into intermittent. It is difficult to determine whether we are not here dealing with endemics of intermittent, which have occasionally made their appearance at the end of the influenza (Biermer). In addition to this, Starck[5] and others have discussed the possibility of an analogy in the poisons of the two diseases.

A connection of influenza with cholera is not probable, as Gluge and Hirsch observe, in spite of the epidemic of 1831 having preceded, and that of 1837 having followed, that disease.

A very widespread epizoötic sometimes occurs among domestic animals, especially among horses, which is designated as "influenza" by the veterinary surgeons. Many of these outbreaks certainly do not belong with this disease, since they may be allied to typhous, erysipelatous, and similar forms of disease not satisfactorily distinguished from influenza. Without regard to the earlier epidemics, however, cording to Woodbury[1] and others, it seems that the pestilence of 1872, which revailed so widely among horses, and which in New York alone attacked about 3,000 of them, was influenza. The disease continued for several weeks, but had aly a slight mortality; in New York 1.5 per cent. No special causes for it were scertained. According to an older account of Hertwig[2] the disease is not inocuble. It is not yet decided whether the epizoötics prevail simultaneously with fluenza among men.

---

[1] Med. and Phys. Jour. Lond., 1803.

[2] *v. d. Busch*, Hufeland's Journ. July, 1834.

[3] *Currie*, Med. and Phys. Jour. Lond., 1803.

[4] Saggio sopra il morbo d. Russo. Vicenza, 1782.

[5] Diss. de catarrh. epidem. 1782. Mogunt, 1784.

PATHOLOGY.

General Course of the Disease.—The influenza is, as Biermer as properly described it, the sum of a series of catarrhal maniestations, which have developed under common epidemic influnces. The intimate association of the various local affections scribed to influenza follows chiefly from their simultaneous ccurrence in large numbers, which allows us to argue a comon origin. Yet, as it seems to me, we do not find a sufficient xplanation of the various diseased processes in the assumption f merely fluxionary hyperæmic lesions affecting the mucous embranes and the nervous system. Many acute local diseases, is true, such, *e.g.*, as bronchial catarrh, angina tonsillaris, etc., un their course not infrequently in a similar way to influenza, ith fever, great sense of illness, and nervous depression. On he other hand, the sudden onset and the often critical terminaion of the disease, its general seizure, the severe nervous sympoms, as well as the decided disposition to cough which invades he organs of respiration, with a proportionately slight increase f the secretion of the mucous membrane, all these are in favor f a general agent which rapidly affects the organism at large. lso many complications which are peculiar to influenza point n the same direction, especially the influence it exercises on xpiring neuralgias, which by it are readily led to relapse, the isposition to abortion, as well as the disturbances which in any cases remain behind long after the subsidence of the fever. hese symptoms are much more severe than in simple catarrhal onditions, while they remind one more of the analogous phenomena in other acute infectious diseases. For these reasons we are surely justified in assigning influenza to this group.

---

[1] Philad. Med. Times, December, 1872, and according to a report in the Lancet of ovember, 1872.

[2] Magazin f. d. ges. Thierheilk. 1854.

From the want of sufficient pathological investigations there still exist many defects in our knowledge of influenza, as in the case of some others of the "great popular diseases," which have hitherto engaged the attention of the historian rather than that of the clinical observer.

The anatomical changes explain very little. Uncomplicated forms of influenza seldom lead to death. As essentially belonging to the disease we find only a more or less considerable hyperæmia and catarrhal swelling of the Schneiderian membrane, as well as of the mucous membranes of the pharynx, larynx, trachea, and bronchi. The catarrh is often limited to the larger bronchi, but in other cases it extends even into their finest ramifications, which sometimes may be filled with clear, thin, frothy mucus, and sometimes with thick, viscid, opaque masses. Catarrhal and croupous pneumonias or pseudo-membranous capillary bronchitis, etc., are included in the complications which generally bring about a fatal result. In the stomach, too, and more rarely in the intestinal mucous membrane, we find more or less extensive hyperæmia.

Symptomatology.

Most frequently the disease is localized upon the mucous membrane of the respiratory organs, more rarely upon that of the stomach and intestines; sometimes nervous accidents are said to exist alone, as Handfield Jones has recently announced, and their relation to influenza is made certain by the epidemic character of the affection. In connection with these we also find various modifications, according to the intensity and the combination of these phenomena.

The *onset* of the disease is generally sudden, as the name "lightning catarrh" indicates. As a rule, it is marked by a decided chill, or by malaise lasting several hours, with slight chills alternating with heat. Only in rare cases are we apt to have malaise for several days, and an established cold beforehand.

In the severer cases the *fever*, even at the beginning, seems attain a rather noticeable intensity. Accurate observations e wanting; according to the older views, it is remittent or sub-ntinuous, and its course is lighter in the day, but in the even-g and at night marked by increase of the various symptoms nd a rise in the frequency of the pulse and heat of the skin. nder some circumstances the temperature occasionally rises ansiently as high as in pneumonia or continued fever.

The *pulse* exhibits a varying character; it may be full and oderately accelerated (90–100), but is also described as small nd weak. It was not infrequently found irregular or very hangeable, so that, as Graves observed, it was often completely anged in from six to eight hours.

Almost invariably a more or less considerable portion of the *ucous membrane*, especially that of the *respiratory organs*, is fected, and this is the characteristic feature of the disease. The mptoms are essentially of a catarrhal nature. We find most equently marked cold in the head, with considerable discharge om the nose and sometimes epistaxis; redness of the conjunc-va, with abundant lachrymation; catarrh of the mucous mem-ane of the throat, larynx, and bronchi; also sore throat, diffi-ilty of swallowing, hoarseness, tendency to cough, and a burn-g or tickling sensation in the throat. The mucous membrane the cavities of the nose, mouth, and pharynx is generally und reddened, and the tonsils swollen.

Häser, and recently Tigri, observed spots like measles on the ucous membrane of the palate, and the latter, in a case which ded fatally, saw also a punctate redness of the mucous mem-rane of the trachea. He regards this phenomenon as just as nstant as the eruption on the skin in exanthematous diseases. iegler[1] infers the existence of a catarrhal œsophagitis, because tients in swallowing warm drinks had a scraping sensation as they had been burned with pepper.

At the very beginning there sets in a very tormenting, convul-ve, *dry cough*, which becomes paroxysmal, especially in the ening and at night, not infrequently leads to vomiting, and

---

[1] Schweizerische Ztschr. 1837, cf. *Biermer*.

and generally the remission of the fever is ushered in by a profuse sweat of acid reaction and strong smell.

In uncomplicated cases the disease generally ends in recovery; its *duration* is three, four, or five days, seldom as much as one to two weeks, and then only as a result of complications. It generally terminates critically, either with sweat or epistaxis, abundant diarrhœal discharges, or increased secretion of urine. In other cases the remission of the symptoms takes place more gradually.

Patients not seldom make but a poor recovery, in spite of the apparently slight affection, so that *convalescence* is protracted. Sometimes, too, there are disturbances left behind, such as headache, muscular debility, cough, etc., which delay the complete restoration to health.

Relapses are not rarely observed, and sometimes occur even in the course of the same epidemic.

These are the most important phenomena of the disease, as described in the majority of cases. But there occur various modifications of this picture in different epidemics, and even among the patients in one epidemic, according to whether the affection is localized more on the mucous membrane of the intestines, or that of the organs of respiration, or according to the increase of the nervous symptoms, which sometimes assume special prominence.

The attempt has many times been made to classify these different forms of influenza according to their special localization. So, *e.g.*, even in the epidemics of 1732–37, there were distinguished an influenza encephalica, thoracica, and abdominalis. Besides these a catarrhal, an inflammatory and a nervous influenza were assumed, a rheumatic-nervous, a nervous-catarrhal, and a gastric form. There were also described, from the peculiar character of the fever, a nervous, a synochal, and an erethetic form.

It is, however, seldom possible to bring all the special cases into such a classification, for the forms under which influenza shows itself are made up of various combinations.

Apart from these, there are, in every epidemic, cases which run a very severe course, and very slight ones, which are to be distinguished from one another. The latter often show themselves only as a simple coryza, laryngitis, bronchitis, etc., but their course is always marked by a material change in the general condition, so that even these patients generally keep their beds.

Finally, we must call attention to the rudimentary forms, a great number of which are observed in every epidemic. A considerable part of the population, in fact, under the influence of the "genius epidemicus," exhibits a state of indisposition which does not amount to a full febrile affection, but which is shown to be a general invasion of the system by slight coryza, by confusion of the head, by one's quickly becoming fatigued, by disinclination for business, and often by sore throat, tickling cough, etc.

The features of the disease acquire still greater diversity from the various *complications*. Many phenomena of the disease, especially the nervous symptoms, exhibit important modifications accordingly as they affect more or less irritable individuals; in hysterical and such other patients influenza often assumes a pronounced nervous or spasmodic character. In the case of hemorrhoidal affections, and in the rheumatic or gouty diathesis especially, the muscular pains will occur. With young children symptoms of congestion of the brain are often seen, and this perhaps may be the explanation of the unfavorable result sometimes observed in their cases.

The influenza process is followed by various sequelæ, and these are so frequent that many observers have indicated them as immediately connected with the essence of the disease; but they may, on the whole, be regarded as merely complications occurring accidentally under specially favorable conditions. Among them are to be counted such cases as present an intense catarrh of the conjunctiva or of the ear, as a concomitant local affection, or also tonsillitis, laryngitis, bronchitis, or pharyngitis of considerable severity. Not infrequently there is left after the recession of the fever a hoarseness or tickling cough, lasting for a considerable time, or a chronic bronchitis or laryngitis. Many patients, too, show a tendency to contract these affections again easily on trifling exposures.

The serious affections of *the lungs* which may accompany influenza are of great significance. Frequently there are only hyperæmia of the lungs and intense bronchitis, but also frequently catarrhal pneumonias, which are to be considered as a continuation of the bronchitis into the vesicles of the lungs. They do not develop before the second or third, generally the fifth or sixth day of the disease, and often slowly and insidiously with the gradual increase of the bronchitic symptoms.

Besides this, croupous pneumonias also occur, and are said especially to involve the period of convalescence.

These complications appear not to be unusual. Among 183 patients received into the Hôtel Dieu, in the first two months of the year 1837, there were forty cases of pneumonia (Copland). Biermer believes that we may assume about five to ten per cent. of the patients as the average frequency of this complication. For some mild epidemics this may be too high; but others, on the contrary, seem to have afforded a larger proportion, at least the blood-letting so frequently employed in former times indicates that the physicians wished to combat inflammatory complications in the lungs.

These pneumonic affections, which have, moreover, a tendency, when the inflammation has once run its course, to establish themselves again in other parts of the lungs, are the cause of the real danger, especially of emphysema, phthisis, etc., which influenza leads to in *the aged* and in persons who are *debilitated* from any other cause. All reports agree in this, that in such patients a fatal result occurs with great frequency, and that the hyperæmia of the rarefied lung-tissue, or the œdema so readily induced by the extensive bronchitic changes, may essentially contribute to it.

Emphysematous and phthisical affections and heart diseases are also considerably aggravated by influenza. Many authors assert, Petit quite recently, that phthisis often runs a rapid course after influenza. It is questionable whether, as many suppose, influenza should be considered as a cause of phthisis. Yet Leudet mentions that after the epidemic of 1851, in Paris, a remarkably large number of cases of acute tuberculosis came under notice.

Pleurisy, without accompanying inflammation of the lungs, appears to be rare. Croup and false croup are sometimes present.

Parotitis with salivation is sometimes observed.

The statements of the earlier observers about the complications of influenza with exanthemata, such as miliaria, roseola, urticaria, petechiæ, scarlatinal spots, erysipelas, etc., seem to refer, as Biermer assumes, chiefly to miliaria and urticaria. Herpes labialis may occur as a favorable indication.

Intermittent fever of tertian type is said to have been developed again and again from influenza.

The effect of influenza upon *neuralgias* is peculiar, since such as have previously got well will often relapse after recovery from this disease. It is also said to leave an unfavorable influence upon other nervous affections.

Pregnant women frequently suffer abortion, and in many who are afflicted with amenorrhœa, the catamenia are established. This effect of influenza has been repeatedly verified in many epidemics. It is therefore very worthy of remark, because it recalls analogous phenomena in other infectious diseases, and is indicative of an agency exerting an important alterative effect upon the whole organism.

Diagnosis.

A positive diagnosis will generally be made with ease from the march of the epidemic, from the great number of persons attacked at the same time, as well as from the relatively severe nervous symptoms, the prostration, and the obstinate cough with but slight physical signs. In the differentiation from non-epidemic catarrh it is essential to make use of the etiological facts at the same time. The catarrhs especially, which occur regularly in changeable weather, in the spring, etc., and which are distinctly influenced by the season, although sometimes marked by severe symptoms, are to be distinguished from the influenza by the fact that epidemics of the latter are positively developed in complete independence of the weather.

Influenza is to be distinguished from a commencing typhoid fever, the early symptoms of which exhibit a certain resemblance to it, by the persistent elevation of the temperature, the absence of enlargement of the spleen and rose spots, the moderately frequent pulse, the peculiar character of the diarrhœa, etc.

Mortality and Prognosis.

In uncomplicated cases the disease commonly ends in recovery. Only certain epidemics have shown a relatively large proportion of mortality; perhaps these reports depend upon the fact that different forms of disease have been erroneously counted as influenza, as has sometimes been the case with typhus fever. Complete and extensive reports on a large number of cases are wanting. Some authors, Gluge, *e.g.*, endeavored to settle the mortality of influenza by inquiring whether the general mortality increased. It appeared, in fact, that in proportion to other periods, the mortality in Berlin, London, Paris, etc., in 1836-7, and also at other times, during the weeks affected by the epidemic, was on an average considerably increased.

There is said to have been shown especially a striking increase of fatal cases due to lung diseases, and among aged persons. Still, the reports differ about this even in more recent epidemics. At all events, it is proved that at a vigorous age the disease is well borne; but that it may easily become dangerous from lung complications in the aged and in persons debilitated by other, especially phthisical, diseases. In young children, too, for the same reason, the disease is not always unimportant.

In *prognosis*, therefore, aside perhaps from the character of the epidemics and the complications, the physical conditions of those attacked are essentially decisive; viz., age, state of strength, existence of other diseases, etc.

Apparently severe cases, too, reach a favorable issue, when they do not affect small children, the aged, the phthisical, or persons otherwise reduced.

TREATMENT.

In the treatment of influenza blood-letting at first played a great part. Yet Buet (cf. Gluge and others) supposes that it is in no respect contraindicated by dyspnœa and bloody sputa, and zanam remarks that in fifty-two epidemics it was useful in hirty-nine, useless in three, and injurious in ten.

Purgatives have been largely recommended, yet have more ecently been advised against (Schweich and others). Emetics ave been much esteemed, and Lombard, of Geneva, still believed n the epidemic of 1837 that an emetic given at the beginning of he disease shortened its course and rendered it milder.

Diaphoretics have been extensively employed; by determina-ion of the blood to the skin they effect a revulsion from the ucous membrane involved. The milder, unstimulating articles specially are chosen, such as ipecacuanha, tartarized antimony, pirit of Mindererus, Dover's powder, packing in wet cloths, etc.

Good results from the employment of quinine were men-oned by Rawling.[1] At all events, further trials of this remedy, large doses, are to be recommended.

In general the treatment will not need to be especially pertur-ting. Quiet rest in bed, and, if necessary, as many think, mild iaphoretic articles (elder or linden-flower tea, etc.) are generally fficient.

An important question in treatment at all times, however, is e control of the adynamia, which must be ranked among the st indications in old people and those who are debilitated. imulants and tonics, especially stimulating expectorants, such senega, camphor, benzoin, solution of ammonia, anise, and the ke, as well as Peruvian bark, wine, etc., are indicated under ch circumstances.

If no contraindication exist, morphine and other narcotics, so an infusion of ipecacuanha, muriate of ammonia, etc., inha-tions of warm steam, or cutaneous irritants of various kinds ay be employed against the annoying cough. Cutaneous irri-nts, or perhaps subcutaneous injections of morphine, inunc-ons of narcotic remedies, etc., are to be prescribed for the relief the oppression of the chest, the pains in the limbs, etc.

Tannin with nux vomica and similar means generally suffice r the more serious diarrhœas; warm fomentations and the ternal administration of narcotics have been employed against e colicky pains.

---

[1] London Med. Gaz., May, 1833.

Oct. 8, Dec. 10.—*Kernig*, Petersb. med. Zeitschr., 1870, 17.—*Thompson*, Brit. Med. J., 1871, Jan. 21, p. 58.—*Fergus*, Ibid., 1871, Jan. 28, p. 90.—*A. Smith*, Med. Press and Circ., 1872, July 17, p. 43.—*Waters*, Brit. Med. J., 1872, Jan. 6, p. 4.—*Stoeber*, Gaz. méd. de Strassb., Févr., 1872.—*Guéneau de Mussy*, Gaz. hebd., 1872. Nos. 1 and 2, pp. 9 and 35.—*Barrat*, Med. Times and Gaz., 1872, June 22.—*Decaisne, M. E.*, De l'Asthme d'été ou fièvre de foin comme entité morbide, Gaz. méd. de Par., 1873, p. 501.—*Blackley, C. H.*, Exper. Researches on the Causes and Nature of Catarrhus Æstivus (Hay Fever or Hay Asthma). Lond., 1873. (Contains the most important experimental and pathological investigations.)—*Morrill Wyman*, Autumnal Catarrh (Hay Fever), with 3 maps. New York, 1872.

INTRODUCTORY REMARKS.

By the name of Hay Fever, or Bostock's Catarrh, is designated a slight febrile affection which attacks a certain, not very large, number of individuals whenever they are exposed to the emanations of grasses in bloom, chiefly just before mowing time. The disease appears in these patients in single accesses which return every year, always running a favorable course, although sometimes with threatening manifestations. The symptoms are, essentially, catarrh of the conjunctiva and of the mucous membrane of the nose and upper air-passages, and frequently, but not always, asthmatic difficulties, which sometimes are of great intensity.

The disease, which is more frequent in England than in other countries, is now referred by Blackley to the operation upon the mucous membranes accessible to it of the pollen of grasses and certain other plants while in bloom (at mowing time).

Little significant as the disease is of itself, it still acquires an increased importance pathologically, because its connection with the noxious influences causing it is established with a certainty common to few other diseases.

The nomenclature of the disease has fluctuated very much according as our ideas of its nature have changed. Bostock named it *catarrhus æstivus*; he wished in this way to indicate its appearance in summer, and, moreover, he regarded the heat of summer as the cause of the disease; still, by this name no distinction is expressed between it and other catarrhs likewise occurring in summer. In England it is called Hay Fever or Hay Asthma. It is also called June Cold, Rye Asthma, etc. Phœbus prefers the name Typical Early Summer Catarrh, as indicating its annual return and the usual time of its accesses; still the disease is not limited to early summer. Biermer proposes, in order to emphasize the element of idiosyncrasy, to make the corresponding addition to the name introduced by Bostock, Summer Catarrh from Idiosyncrasy. Blackley finally, from his investigations, recommends the names Pollen Catarrh and Pollen Asthma, on etiological grounds. The name "Hay Fever," however, has become most widely known, and this may still be retained, unless it be preferred to call the affection, from its first observer, Bostock's Catarrh.

Our knowledge of this disease was first founded in the year 1819 by Bostock, who was himself attacked by it, and first gained a knowledge of twenty-eight cases (observed by himself and communicated to him) after the publication of the history of his own disease. He cites a statement of Heberden's,[1] according to which the latter had observed before him some undoubted cases of summer catarrh; and Phœbus believes him to have been the only author acquainted with the disease before Bostock. Still, Biermer adduces one passage also from van Helmont[2] and from Bosquillon,[3] which probably refer to this affection. Other citations are not free from question.

Up to the year 1861, only a small number of works (twenty) had appeared on this subject, and these Phœbus collates. It was only in England, however, that attention to this disease was extensively aroused; upon the Continent, and especially in Germany, it was little heeded, as proved by the absence of this heading in most of the handbooks. In 1854 Phœbus was induced from special motives to collect extensive material for observation; he therefore issued a circular to the medical societies, etc., by means of which he succeeded in obtaining notes of 154 cases, the larger part of which had never been published, and these he carefully analyzed. His book contributed essentially to arouse an interest in hay fever.

Since the appearance of Phoebus's work there have followed quickly, one after another, a large number of publications (in part cited by Biermer, and to the latest period by Blackley).

---

[1] I have known it (catarrh) return in four or five persons annually in the month of April, May, June, or July, and last a month, with great violence.

[2] Vidi frequenter mulieres, quæ suavi olentium odore præter cephalalgias et syncopes, confestim in extremam respirandi difficultatem inciderent.

[3] *Bosquillon* relates (conf. Cullen's First Principles of Pract. Med.) that he has known a strong full-blooded man who always had an asthmatic attack when rice was threshed in the neighborhood of his dwelling.

The case of Helmholtz (by Binz) is of especial interest, for he himself was attacked by hay fever for several successive years, and referred it to vibriones which were found in the nasal mucus.

The number of cases which have come under observation Phoebus estimated as altogether about 300. Since then, perhaps about 150 more may have been recognized.

Very recently there has appeared on this subject the highly important work of Blackley, who, himself a sufferer from hay fever, has kept up experimental studies in it for ten years. The result of his labors is extremely important, because it puts beyond question the complete dependence of the disease upon definite causes.

ETIOLOGY.

In considering the *etiology* of Bostock's catarrh, we must, more than in the case of almost any other disease, draw a sharp distinction between the predisposing and the exciting causes; for it must be strikingly apparent throughout that this affection is exclusively confined to certain individuals, in whom, therefore, certain definite predisposing influences are to be assumed. With these persons the disease appears year in and year out, in more or less regular accesses, often under the most different external conditions.

It has been made evident by the compilation of Phœbus and by Blackley that the disease only attacks individuals under forty years of age; up to that, however, no age escapes. The youngest patient among fifty-six cases in Phœbus, was five and a quarter years old, and among sixteen cases in Blackley four years. The majority of cases of the former, viz., eleven, come in the class from sixteen to twenty years, and the next greatest number, viz., ten, in that from six to ten years old.

In regard to *sex*, the female is out of proportion less frequently affected than the male; 104 male to 50 female patients (Phœbus). The ratio is similar in more recent authors. It is proved, at any rate, that the hysterical impulse, to which some authors attribute great influence, can play no important part among the predisposing causes.

The patients belong exclusively to *the educated classes;* the clergy, officers, physicians, merchants, and their relations. The number of physicians is relatively the most largely represented, perhaps because they pay most attention to the disease. It is particularly noticeable that no person living in the country is found among the patients. Blackley concludes from this, as well as from the larger number of cases published very recently, as opposed to the wholly isolated cases of the previous century, that the ever-increasing discontinuance of rural occupations and the longer stay in closed rooms, occasioned by social conditions, have constantly increased the predisposition, and that this tendency will probably be still further developed in the future.

All *constitutions* and temperaments, but particularly the nervous, are liable to the disease. It appears from this, too, as Phœbus supposes, that *hereditary tendency* plays a certain part in the predisposition.

To this must be added, that some patients at other seasons of the year, independently of hay fever, suffer from urticaria.

The disease is known as especially prevalent in England proper. Of the 154 cases of Phœbus, 83 came from England, 34 from Germany, 16 from France, 7 from Belgium, 3 from Switzerland, *only* 2 *from Scotland,* 3 from Italy, and the rest from other countries. The more recent authors report that the hay fever is not very rare in America also. Blackley knows of some persons from India. Some patients, moreover, suffer their annual attacks, even though far from home (Phœbus).

Among the *exciting causes* are to be mentioned only the influence of the *seasons,* and the *blossoming period of many grasses.*

The disease occurs only in early summer and midsummer, except under certain circumstances in the latter part of summer too, viz., up to the end of the second mowing; in England in the months of May, June, and July, seldom up to September. The annual attacks keep invariably to these seasons; those who have repeatedly been visited by it of course fear its recurrence, which they confidently expect, and in consequence the nervous excitability in many reaches a high degree even before the time.

The patients uniformly assert that the affection is not caused by *atmospheric conditions*, but by certain *vegetable emanations*. As soon as the patient approaches a meadow in full bloom, or a field of rye, flowering grass or the like, or a heap of freshly mown hay, he is attacked by the disease. A female patient (Phœbus) related that she brought on her attack in two successive years by receiving a bunch of grasses and wild flowers.

These statements have from the beginning directed attention to the emanations proceeding from growing plants. Some even assume that the efficient cause is to be sought only in the high temperature which prevails at the same time, etc. The following influences may therefore be assigned as causes of the disease.

1. The influence of dust; 2. The odors of various kinds of flowers, especially the ethereal oils which go along with them; 3. The cumarin which occurs in certain plants, and especially in those grasses under consideration, such as anthoxanthum odoratum (sweet-scented grass), and which is also developed perhaps in the hierochloa and melilotus varieties in the fresh, but more decidedly in the dry state; 4. Benzoic acid, which is the source of the aroma in anthoxanth. odor., holcus odor., etc.; 5. The influence of ozone; 6. The effect of sunlight and heat; and 7. The operation of the pollen of grasses diffused in the air.

Many patients have set on foot experiments in these directions. Blackley carried them on systematically in such a way as to study the influence of the various substances alluded to upon his respiratory mucous membrane His results are the following: a thorough observation of atmospheric conditions showed that the attacks do not run at all parallel with extremes of light and heat. Ozone, benzoic acid, and cumarin produced only a very unimportant influence upon the respiratory organs, and none which resembled the symptoms of hay fever. Dust, which besides is differently constituted in different regions, may occasion cough and catarrhal symptoms at various seasons: but these have little similarity to hay fever, and are not at all confined to the summer season. The fragrance of flowers of various kinds has little effect, although the smell of chamomilla matricaria produces disagreeable symptoms, headache, etc. The effect of the spores of penicillium glaucum was hoarseness increasing to aphonia, bronchial catarrh, etc., which lasted for some days.

On the other hand, there has been complete success in developing the features of hay fever by the operation of pollen upon the organs of respiration. Blackley experimented with the pollen of seventy-four kinds of plants, among them many grasses. The investigations were undertaken at different seasons of the year with dried and fresh flowers. The great majority of the pollens gave like results.

The grains of pollen present a simple cell formed of an external and internal membrane, which is filled with granular contents. The membrane often bursts, and the latter become free. The absorption of water effects a speedy bursting, with vigorous dispersion of the granules; this takes place somewhat more slowly from the action of mucosities and other substances. The granules, when colored with aniline and carmine, frequently exhibit an active molecular motion, lasting for a considerable time.

The mechanical operation of the grains of pollen upon the mucous membranes probably depends upon these circumstances. Besides this, they must have a chemical effect, too, a view which has not yet been closely investigated, but which certain experiments favor. The mode of operation of the pollen grains is also somewhat altered by oil, etc. Dried pollen can be vitalized again by the absorption of water.

The influence of the pollen upon the mucous membranes was investigated in the following way: If grains of the pollen of *lolium italicum*, *e.g.*,were placed upon the mucous membrane of the nose with the tip of the finger, after a few hours the symptoms of a cold in the head appeared, completely corresponding with what is observed in hay fever. When a larger quantity was used, there resulted the characteristic violent sneezing. The inhalation of pollen through the mouth produced asthmatic symptoms, with general illness. A drop of a one per cent. decoction of the pollen of *gladiolus*, laid upon the conjunctiva, immediately excited a conjunctivitis, which lasted for thirty-two hours.

Also, the inoculation of the pollen under the skin gave evidence of its irritating properties by inflammatory swelling of the affected part.

At any rate, these experiments, which may be followed out in detail in the original, prove that the pollen, as well in the fresh as in the dried state, is capable of producing all of the symptoms of hay fever.

A further series of observations served to establish the relative quantity of pollen present in the air, and its relation to the intensity of the symptoms of the disease. Small plates of glass, covered with a viscid fluid (water and glycerine, each 100 parts; alcohol, 200; carbolic acid, 2.5 parts) were exposed to the air in a suitable apparatus, and the pollen grains collected in a certain time upon a square centimetre counted. Observations, continued for several months, gave, at a mean temperature of 23.5° C., an average of about 364 grains of pollen to one square centimetre in a day. The observations were continued for two years, 1866 and 1867; in the former the mean number amounted to 472.5, in the latter, to only 46.8 pollen grains in a day. The maximum, 880, was attained on June 28; the highest temperature in that year, 35.6° C., was observed on the day before. In the second year the greatest quantity fell upon June 23. In both cases the number of the pollen grains diminished continually on the one hand up to July 28, and on the other to May 28, within which period the observations were carried on.

The maximum numbers given coincided in the city and country. In large cities the number of the pollen grains was disproportionately less than in the country, on an average in about the ratio of 1 : 4.4. On June 28, 1866, the number for the country amounted to 880; for the city to only 104.

Besides this, the observations showed that in the more elevated strata of air the pollen grains are disproportionately more numerous than in the lower ones, while we should have expected the reverse condition. For a considerable difference of height, the relation of the places situated at a higher level (1,500′) to the lower is placed as 104 : 10.

Rainy weather notably diminishes the quantity of pollen, and the strength and direction of the wind have also a great influence upon it.

In conformity with these observations, it is shown that the greatest intensity of the symptoms of hay fever in the case of

Blackley himself always agreed with the maximum amount of pollen diffused in the air. The attacks are more violent in the country than in the city, and regularly subside more or less suddenly on the occurrence of rainy weather,—observations which entirely coincide with the statements of the authors above quoted.

Further experiments show the reason, too, why all the symptoms of hay fever are worse when the patient moves about actively, but diminish when he keeps quiet. Without considering the fact that we breathe more frequently when in motion, 4.4 times as much pollen is deposited upon the same surfaces with a motion of two English miles an hour as when at rest (in the one case, *e.g.*, 140, in the other 28, in thirty minutes, on one square centimetre).

For the same reason probably the attacks are milder in-doors than in the open air. When in good seasons there is a rapid growth of grass, and haymaking is favored by the weather, so that two mowing times follow closely one upon the other, it may happen that the attacks of persons dwelling in such districts are unusually prolonged, so as to last from May to September; while, under the contrary circumstances, they run a much shorter course.

Symptomatology.

Blackley makes use of the results arrived at to explain the features of the disease, the symptoms of which have necessarily seemed exceedingly paradoxical, and have called forth the most various hypotheses.

Phœbus arranged the symptoms in six groups; symptoms referable to the nose, to the eyes, to the throat and mouth, to the head, to the neck and chest, and the general phenomena. Still it is sufficient to make an essential distinction of two classes, between which all the manifestations may easily be represented; the catarrhal form and the asthmatic.

Patients may be attacked with both of these forms, or with either one of them alone; sometimes it amounts merely to a local affection of one portion of a mucous membrane.

The *catarrhal form* runs its course with little pain and no important symptoms, while the asthmatic sometimes occasions very alarming manifestations, but like the former always ends favorably.

According to Phœbus, the disease often begins with prodromata of several days' duration, with weakness, coated tongue, diarrhœa alternating with constipation, sleeplessness, etc. Sometimes it begins after only a few hours' discomfort, or else suddenly. Blackley observed only the latter variety. Records of the temperature, taken at two periods during the time from April 28 to June 28, showed no elevation of the animal heat up to immediately before the beginning of the attack, and no acceleration of the pulse. If previous indisposition exist, it is probably generally unconnected with hay fever. That is to say, it is quite possible that one and the same amount of pollen bodies in the air will be well borne by one patient from his individual capacity for irritation, while it causes lesions in another.

The affection attacks exclusively, and indeed in the following order, the nasal mucous membrane, the conjunctiva, the mucous membrane of the mouth and the fauces, and that of the larynx, the trachea, and the bronchi. When the wind is strong, the conjunctiva is often seized first; under such circumstances, the wind is often regarded as the cause; while, according to Blackley, this should rather be sought in the increase of pollen it brings with it.

The first symptom, just as in the experiments already mentioned, is a rather marked tickling. Then a clear serous fluid begins to flow from the nose, and violent sneezing occurs, often twenty or thirty times, one after another, and even more; lachrymation also occurs sometimes. The nasal mucous membrane swells up more or less quickly, according to the amount of the pollen affecting it. Not rarely the swelling is so great that the two nostrils are closed. If the patient lies on one side, the swelling goes down, since the œdematous effusion is easily movable, and gravitates.

So soon as the Schneiderian membrane is highly swollen, it becomes less sensitive, even to the pollen, which, as stated, acts as an active sternutatory, and the sneezing ceases. At times, slight bleeding occurs, from which the nasal mucus appears of a rusty tinge, as in pneumonia. Later on, it becomes thicker, and ultimately even purulent, but not to a high degree. The shedding of epithelium seems to be less considerable than in ordinary coryza.

The *eyes* appear to be affected somewhat later, because the number of pollen grains is smaller in proportion to the fluid which washes them away, than in the case of the nose. Still the affection sometimes even begins here. The symptoms here are similar to those in the case of the nose,—tickling, which increases to more decided burning, swelling, and redness of the conjunctiva, as well as of the anterior portion of the globe. The tear passages are sometimes completely closed by swelling. Shooting neuralgic pains are felt in the posterior portion of the orbits. When very many pollen grains are present, a slight chemosis is established. Sometimes the patient presents photophobia, which, however, is not very intense, and even allowed Blackley to use his microscope. In the morning there is œdematous swelling of the eyelids. The secretion is at first thin and watery, then grows thicker, and may even become purulent.

The mucous membrane of the *fauces* and *mouth* is less sensitive; it becomes swollen, to be sure, but not markedly. In the pharynx the patients are conscious of a tickling, a sense of dryness and pressure, or, later, a slight burning feeling, which is perceived as far as the Eustachian tube and the meatus auditorius externus. Sometimes also there occurs a slight deafness, and this constitutes the only symptom which may sometimes last for a considerable time after the attack. Rarely there exists a slight cough. Shooting pains in the head probably have the same origin as in the case of the eyes.

The *asthmatic form*, in the course of which alarming symptoms sometimes occur, is constantly accompanied by laryngobronchial catarrh. The asthma is of varying severity, from a moderate cough, with scanty expectoration and a feeling of constriction about the chest, to great dyspnœa, croupy inspirations, a sense of anxiety, and a pale and disturbed expression of countenance.

In the most severe cases the sense of suffocation is very considerable. The muscles of respiration act with the greatest force, the patient sits upright, with the hands convulsively clenched, and is extremely anxious.

As a point of distinction from nervous asthma, the attacks of hay fever will be found to occur mostly in the daytime, in the fresh air; and, while they show brief remissions, they still have a tendency to last with greater or less severity during the whole hay-fever season. In one case the patient had to spend twelve or fourteen nights in this condition, sitting up. (She was in the country at the time, while usually she lived in a suburb of Manchester.) Blackley refers this affection also to the action of pollen grains, which often burst as soon as they become moist, and discharge their granular contents upon the mucous membrane. It is proved by the experiments that they quickly cause a considerable hyperæmia or inflammation, and œdematous transudation in the mucous membrane. The proof that they are retained here in large amount is found in the fact that the pollen is, out of proportion, less abundant in the expired air than in that which is inhaled.

But the high degree of functional stenosis caused in this way would be sufficient to explain the asthma. The inhalation, too, of pollen, undertaken for experimental reasons, induced the same symptoms.

The general symptoms are not characteristic; some patients are disinclined to work, being easily exhausted; some are prevented from sleeping by a rapid stream of thoughts. Sometimes neuralgic pains occur, similar to those which are perceived in the thumb and forefinger on the inoculation of pollen under the skin of the arm. The fever is seldom considerable, except in asthma; the pulse in that case becomes frequent and full, the skin hot and dry, and there are chills from time to time.

For reasons which are easily perceived, these symptoms seldom occur simultaneously; generally only one group or another is specially represented. They last for three or four weeks, with more or less noticeable intermissions, and do not lead to serious lesions, but, as a rule, vanish with striking rapidity, perhaps in favorable weather completely. The asthma disappears as with a blow, and the mucous membranes of the nose, the eye, the mouth, the pharynx, etc., return to their normal state in a few hours, after a night, or at most in from one to one and a half days.

Whether the course of the disease is of greater or less intensity and duration depends, according to Blackley, upon the amount of pollen distributed in the air, and upon the greater or less sensitiveness of the patients.

Under some circumstances the attacks may extend over a space of three months.

Patients are generally visited by these seizures every year. The earlier attacks are, as a rule, milder than the later ones. Sometimes, too, under the circumstances mentioned above, there occur secondary accesses at the time of the second mowing.

Diagnosis.

With regard to *diagnosis*, our only concern is really with the distinction of the affection from sporadic catarrhs, and this depends on our knowledge of the accesses as annually recurring at the regular season. The attacks are always most violent during a stay in the vicinity of blossoming meadows, etc.

Besides this, hay fever is to be distinguished from those catarrhal attacks which are due to the inhalation of the dust of powdered ipecacuanha, and the emanations of oil of turpentine, of certain kinds of pine, etc., but which are always merely accidental and transient. (Phœbus adduces a longer list of such instances.)

TREATMENT.

Treatment is still powerless against hay fever. Phœbus cites a long list of remedies which have been tried in vain. The injection of a solution of quinine into the nostrils, as employed by Helmholtz, has proved ineffectual in subsequent cases.

Change of air does not always protect one from the attacks. Even a stay at the sea-shore is not alone sufficient, for the attacks return as soon as land-winds blow. Sea-voyages alone furnish immunity.

The English physicians often send their patients to the north of Scotland, where the meadows are not in bloom at the time of hay-cutting in England. In America, they are in the habit of resorting to an island named Fire Island, a little separated from the mainland, near Long Island, where they remain free from the disease during their stay.

After all, then, the result of the researches of Blackley is, that patients had better remain in the city in summer, and as much as possible in their rooms, where, at all events, they will have to endure less severe seizures than in the open air.

It is impossible constantly to avoid the noxious influences. No means of eradicating the tendency to the disease by a process of hardening, or the like, has yet been found.

MALARIAL DISEASES.

HERTZ.

MALARIAL DISEASES.

---

nac, De revoud. febr. interm. etc. 1759.—*Coling*, De febrib. intermit. 1760.—*Medicus*, Sammlung u. Beobachtungen. Zürich, 1764, and his Geschichte, etc. Frankf., 1795.—*Pringle*, Beobachtungen, a. d. Engl. von *Braude*. Altenb., 1772.—*Lind*, Ueber die Krankheiten der Europäer in heissen Klimaten. Riga, 1773.—*Trnka de Krzowitz*, Hist. febr. interm. 1775.—*Strack*, Obser. med. de feb. interm. Offenb., 1785.—*Grainger*, Prakt. Bemerkungen. Leipzig, 1785.—*Stoll*, Aphorismen, etc. Vendob., 1785.—*Thomson*, Treatise. London, 1787.—*von Hoven*, Das Wechselfieber und seine Behandlung. Winterthur, 1789.—*Jackson*, Ueber die Fieber in Jamaica, Uebers. von K. Sprengel. Leipzig, 1796.—*Balfour*, In various publications.—*Dawson*, Observat. on the Walcheren Diseas., 1810.—*Davies*, A Scient. and Pop. View, etc., 1810.—*Wright*, Hist. of the Walcheren remit. 1812.—*Audouard*, Nouv. thérap. de fièvres intern. Paris, 1812.—*Sebastian*, Ueber die Sumpfwechselfieber in Holland. Karlsruhe, 1815.—*Alibert*, Traité de fièvr. pernicieus. interm. 5. édit. Paris, 1820.—*Monfalcon*, Histoire des marais. Paris, 1824. Deutsch von Heyfelder. Leipzig, 1825.—*Bailly*, Traité anatom. patholog. de fièv. intermit. Paris, 1825.—*Bakker*, De volksziekte van het jaar 1826.—*Buchner*, Verhandeling over den invloed der Nord-Hollandsche Droogmakerry. Utrecht, 1826.—*Thyssen*, Over de herfstkoortsen te Amsterdam, 1827.—*Thussinnk*, Algemeene overzicht, etc. Groningen, 1827.—*MacCullock*, Malaria, etc. Lond., 1827.—*v. Reider*, Untersuchungen üb. d. epidemischen Sumpffieber. Leip., 1829.—*Boyle*, An Account of the Western Coast of Africa, etc. Lond., 1831.—Verhandelingen over de epidemische ziekte in de Nederlanden 1831.—*Hasper*, Krankh. d. Tropenländer. Leip., 1831. 2. Th., p. 187.—*Nepple*, Sur les fièvres rémit. et intermit. Par., 1835.—*Maillot*, Traité des fièvres intermit. Par., 1836.—*Kremers*, Beobacht. üb. d. Wechself. Aachen, 1837.—*Monyellaz*, Monographie des irritat. interm. Paris, 1839.—*van Geuns*, Natuur- en geneeskundige beschouwingen van moerassen en moerasziekten. Amst., 1839.—*Eisenmann*, D. Krankheitsfamilie Typhus. Zürich, 1839.—*Savi*, Sull cativaria delle maremme. Pisa, 1839.—*Shewardson*, Amer. Jour., 1841, April.—*Molo*, Ueber Epid. u. Wechselfieber-Epidemien, etc. Regensb., 1841.—*Boudin*, Traité des fièvres interm. Paris, 1842, and Essai de géograph. médic. Paris, 1842.—*Fergusson*, Edinb. Jour., Vols. 59 and 60. Medic. Jahrbücher f. d. Herzogthum Nassau, 1843.—*Robertson*, Med. Notes on Syria. Edinb. J., Januar.,1843, Vol. 60. *Eisenmann*, Med.-chir. Zeitsch., 1843, No. 17.—*Piorry*, Comptes rendus, T. XVI.

No. 3., Gaz. des hôpit. 1844, No. 45. Traité de médec. pratique, etc., 1845. Tom. VI.—*Steifensand*, Casper's Wochenschr. 1853, No. 44.—*Dietl*, Zeitsch. f. wiener Aerzte, 1844, Decbr.—*Wilson*, Edinb. Jour., 1846, Vol. 66.—*Jacquot*, Gaz. médic., 1848.—*Heusinger*, Recherch. de pathol. comp. I.—*Steifensand*, Das Malariasiechthum in den niederrhein. Landen. Crefeld, 1848.—*Pfeufer*, Zeitschr. für ration. Medicin, 1849, 1 and 2. Heft.—*Canstatt*, Prager Vierteljahrschr., 1850, Bd. 4.—*Wolff*, Annalen des Charité-Krankenhauses, 1850, 1.—*Grisolle*, Gaz. des hôpit., 1850, No. 65.—*van Deen*, Nieuw Archief., Bd. III, p. 305.—*Heschl*, Zeitschr. für wiener Aerzte, 1850, Juli.—*Heinrich*, Med. Zeitung Russlands, 1850.—*Meckel*, Deutsche Klinik, 1850.—*Rineker*, Verhandlung der physik.-med. Gesellschaft in Würzburg, 1851.—*Dundas*, Sketches London, 1852.—*Dietl*, Oester. med. Wochenschr., 1852.—*Haspel*, Maladies de l'Algérie. 1852. Vol. 2.—*Epp*, Schilderungen aus holländisch Indien. Heidelb., 1852.—*Panum*, Verhandlg. der würzb. Gesellsch. 1852.—*Bonnet*, Traité de fièvres intermit. Paris, 1853.—*Bierbaum*, Das Malariasiechthum. Wesel, 1853.—*Clemens*, Zeitschr. für Staatsarzneikunde. 1853.—*Planer*, Zeitschr. der wiener Aerzte. 1854.—*Zimmermann*, Clin. Untersuchung. zur Fieber-Entzündungs- u. Krisenlehre. Hamm, 1854—*Jacquot*, Annal. d'Hygiène publiq., 1854, 1855.—*Frerichs*, Die Melanaemie, etc., Zeitschr. f. clinisch. Medic., Breslau, 1855.—*Jones*, Assoc. Med. Jour., 1856, Aug.—*Michael*, Special-Beobachtungen der Körper-Temperatur im intermittirenden Fieber, Archiv für physiol. Heilkunde, 1856. Heft 1. p. 39.—*Griesinger*, Infectionskrankheiten, in Virchow's Hdbch. der spec. Path. u. Ther., 1857 and 1864, Bd. II, Abth. 2.—*Mouchet*, Revue médic., 1857, April, Mai.—*Scholz*, Zeitschr. d. wiener Aerzte. 1857, Mai u. Juni.—*Balfour*, *Baikie*, Edinb. Med. Jour., 1857, March.—*Hirsch*, Klinische Fragmente. Königsberg. 1857.—*Heidenhain*, Virchow's Archiv, Bd. XIV, p. 509.—*Redenbacher*, Harnstoffgehalt des Urins, Henle und Pfeufer's Zeitschrift, III Reihe, 3 Band.—*Rigler*, Wien. med. Wochenschr., 1858, 8.—*Duchek*, Prager Vierteljahrschr., 1858, 4, Spitalszeitung, 1859, 12–20.—*Hirsch*, Handbuch der historisch-geographischen Pathologie. Erlangen, 1859. Bd. I, p. 5.—*Ringer*, Lancet, 1859, Aug. 6.—*Frerichs*, Klinik der Leberkrankheit, Chapter on Melanæmia.—*Jones*, Observat. on some of the Physical, Chemical, Physiol., etc. Philadelphia, 1859.—*Oppolzer*, Wien. med. Wochenschr., 1860, Nr. 25, 26.—*Duboué*, Moniteur des sc. méd., 1861, Nr. 83 et seq.—*Mayer*, Würtemb. Correspondenz-Bl., 1861, No. 34.—*Heschl*, Oester. Zeitschr. f. prakt. Heilkunde, 1872, Nr. 40–43.—*Key*, Prager Vierteljahrschr., 1862, Bd. III.—*Friedmann*, Deutsche Klinik, 1863, No. 1.—*Saint-Vel*, Gaz. heb., 1863, No. 13.—*Rosenstein*, Allg. wien. med. Zeitung, 1864, Nr. 41.—*Ritter*, Virchow's Archiv, Bd. XXX, p. 273, Bd. XXXIX, p. 14, Bd. XL, p. 239, Bd. XLVI, p. 316, Bd. L, p. 164.—*Boullet*, Sur les causes des fièvres intermit. Thèse de Paris, 1864.—*Baxa*, Wien. med. Wochenschr., 1866, 78.—*Thomas*, Archiv f. Heilkunde, Bd. VII, pp. 225, 289, and 385.—*Salisbury*, Amer. Journ. of Med. Sc., 1866, Januar.—*Schwalbe*, Archiv f. Heilkunde, 1867, Heft 6, p. 567.—*Colin*, Union méd., 1867, Nr. 118 et seq.—*Jilek*, Ueber die Ursachen der Malaria in Pola. Wien, 1868.—*v. Frantzius*, Virchow's Archiv, Bd. 43, p.

815.—*Rühle*, Med. klin. Wochenschr., 1868, Nr. 10.—*Barat*, Archiv. de Méd., 1869, Dec., 422.—*Barraut*, Compt. rend., 1869.—*Schwalbe*, Beiträge zur Kenntniss der Malaria-Krankh. Zürich, 1869.—*Lorinser*, Eucalyptus glob., Wien. med Wochenschr., 1869, Nr. 43, 1870, 27.—*Colin*, Arch. gén. de méd., 1870 Jan., p. 5, and Traité de fièvres intermit. Paris, 1870. Gaz. heb. de méd., 1872, Nr. 35, p. 563. Annal. d'hyg., 1872, Oct., p. 211. Annal. d'hyg. publ. et de méd. lég., 1872, p. 241-76.—*Wenzel*, Prager Vierteljahrschr., 1870, IV, p. 1, and Die Wechselfieber in ihren ursächl. Beziehungen während des Hafenbaues im Jadegebiet. Prag., 1871.—*Tessier*, Brit. Med. Jour., 1870, Dec. 31.—*Blaxall*, Med. Times and Gaz., 1870, Jan. 8, p. 49.—*Treulich*, Wien. med Presse, 1871, Nr. 12.—*Lacaze*, Union méd., 1872, Nr. 116.—*Bérengar*, Gaz. des hôp., 1872, p. 145.—The literature of eucalyptus globulus during the year 1872: *Stube*, Berl. klin. Wochenschr., No. 52.—*Keller*, Wien. med. Wochenschr., p. 10.—*Mosler*, Deutsches Archiv, X, p. 159.—*Castan*, Montpellier méd., Mai, p. 385.—*Papillon* Gaz. hebd. de méd., No. 31, p. 501.—*Bertheraud*, Gaz. méd. d'Alger., Nr. 12.—*Armand*, Traité de climatologie génerale du globe, études méd. sur tous les climat. Paris, 1873.—*Curschmann*, Behandlung des Wechself., Med. Centralbl, 1873, p. 628, and Deutsches Archiv, 1872, Bd. IX.—*Fiechter*, Ueber die Wirkung der Tinct. Eucalyp. glob., Deutsches Archiv, Bd. XII, Heft 5, p. 508.—*Mees*, Over werking van Eucal. Glob. Dissert. inaug. Groningen, 1873.—*Mosler*, Wirkung des kalten Wassers auf die Milz. Virchow's Archiv, Bd. 57, p. 1.—*Henoch*, Febr. intermit. perniciosa. Berl. klinische Wochenschr., 1873, Nr. 26, p. 301, and Nr. 34, p. 402.—*Eisenlohr*, Carbolsäure gegen Intermittens ebend. 1873, Nr. 41, p. 487.—See, in addition, the literature in *Virchow* and *Hirsch's* Jahresbericht and *Schmidt's* Jahrbücher.

HISTORY.

THE history of this disease, in all its various types, reaches back to the earliest period of medical science. Not only were the common forms then known, but also, and particularly, the uncommon and pernicious varieties of the malady. Protagoras describes the drowsiness accompanying intermittent fever, and the tetanus which sometimes supervenes, and tells of many cases that terminated fatally, whence it would appear that he may have been the first to observe pernicious intermittent fever (febris int. comitata). Celsus draws the distinction between quotidian, tertian, and quartan fever, and refers to the possibility of a longer intermission in these words:—"Interdum etiam longiore circuitû quædam redeunt, sed id raro evenit." He also speaks of the genus *ἡμιτριταῖον*, which he describes as a fever lasting three days, with uncommonly long paroxysms reaching over into one another. (Lib. III, cap. 3). Archigenes was the first writer who recognized the complex nature of the hemitritæa, or semi-tertian form, as consisting of a tertian and a quotidian fever combined. He also makes mention of masked intermittents, especially when appearing in the form of dysentery or diabetes. Rhazes, the Arabian, gives an account of febris subintrans. Ebn Sina saw that rare type in which the fever occurs every sixth or seventh day, and Valescus, of Taranta, refers to a fever recurring every thirtieth day. (Philos. pharmaceut. et chirurg. lib. V. ed Hartmann Beyer, Francf., 1599. Lib. VII, p. 596). Rembert Dodöeus (Méd. observat. exempl., C. 4, p. 9), describes that form of intermittent fever designated as "katochus," and Diomedes Cornarus (Com. Lips. 1599, p. 28), first noticed combinations of intermittent fever with dysentery, or rather the intermittent type of dysentery.

It was not, however, until after the introduction of cinchona bark from Peru into Spain, in the year 1640, by the Viceroy del Cinchon and his body-physician, Juan del Vego, that any considerable degree of attention was given to the study of intermittent fever. This study was now stimulated in part by the conflicting results following the use of the new drug, and in part by the obstinate manner in which physicians generally clung to the old theories of Galen. No truly reliable results were reached in practice until after the apothecary's clerk, Robert Talbor, or Tabor, of Cambridge,[1] had introduced the use of larger doses and more effective forms of administration (mostly macerations in wine, or the tincture, with or without opium, which preparation he sold extensively as a secret remedy for ague); and until Sydenham conceived the idea of giving the cinchona immediately after the first attack, during the intermission, for the purpose of forestalling a subsequent paroxysm. Hereupon followed the classical work of Torti (Therapeutice specialis ad febres quasdam perniciosas. Mutin., 1712), in which the true nature of pernicious intermittent fevers is recognized and their treatment given; and the work of Lancisi, which is no less valuable, especially from an etiological point of view (De noxiis paludum effluviis eorumque remediis. Genève, 1716). The labors of Guisman Galeazzi and of Werlhof (Observat. de febrib., præcipue intermitt. Hannover, 1732) followed in the same direction as those of Torti.

---

[1] Pyretology; or, A Rational Account of the Cause and Cure of Agues, etc. London, 1672.

Malarial affections comprise a class of diseases that are usually marked by a perceptible, often a very considerable, rise of temperature, and which are furthermore to be regarded as constituting different forms of one and the same morbid process, because of their origin in the same purely miasmatic causes, the fact of their frequently occurring simultaneously within the same area, the nature of their anatomical products, as well as certain peculiarities characteristic of their course. In addition to this, they frequently run into one another, and they all yield to pretty nearly the same remedies. They may be divided into, 1st, simple, benign intermittents; 2d, anomalous or masked intermittents; 3d, pernicious intermittents; 4th, remittent and continuous fevers; and 5th, malarial cachexia.

GEOGRAPHICAL DISTRIBUTION.

There is probably no part of the torrid or temperate zones in which greater or smaller areas of country may not be found where malaria is endemic, and where it prevails every year to a greater or less extent. Although it is foreign to the purpose of the present work to enter upon a minute investigation of this matter, yet we may be permitted here to mention the most important foci of malarial infection.[1]

Europe.—One of the most famous malarial regions of *Germany* lies south of the Carpathian mountains, embracing the major part of Hungary, what are called the greater and lesser Hungarian plains, Banat, Croatia, and a part of Slavonia. Not less extensive and notorious are the fevers of Dalmatia and Istria, while they prevail under a milder form in the great plain of the Danube in Lower Austria and in the marshes, as well as in the northern portion of Galicia. A second very extensive malarial region exists in the North-German flatlands: Northern

---

[1] A more complete account may be found in *Hirsch*, Handbuch der historisch-geographischen Pathologie, Bd. II, p. 5, whom, in the main, we have followed.

Silesia, the plain of the River Mark, the Baltic coast of Prussia, Pomerania, and Mecklenburg; the marsh and meadow regions of Hannover and Oldenburg; strips of the western coast of Holland and Schleswig; the lowlands of Westphalia, as well as the marshy plains of the Rhine and its tributaries, including Holland, especially its seaboard provinces of Gröningen, Friesland, North and South Holland, and the long-famed Zealand; furthermore, the northern and western provinces of Belgium, particularly those of West Flanders and Antwerp.

In the mountainous regions of Upper Austria, in Tyrol, Carinthia, Steiermark, Bohemia, and Moravia, and in the hilly country of middle and south-western Germany, malaria is entirely unknown, or occurs only in single localities (as in Rhinegau and the Danube bottom-lands of Würtemberg and Bavaria). Switzerland is also free from it, except on the margin of some of its lakes, as of Lake Zürich and Lake Constance.

In Denmark malaria is endemic only on the humid islands of Falster and Laaland. In Sweden it is to be found on the coast of the Baltic, in the districts of Carlskrona, Södermanland and Gestrikland, on the banks of the River Angermann (62° 20′ N. lat., the most northerly limit of the disease in Europe), and on the shores of the three great lakes, Maelar, Wener, and Wetter.

In Norway, Iceland, and the Faroë Islands, the disease does not seem to exist at present.

In Russia it may be found in the provinces bordering on the Baltic, especially in Esthonia; also in Litthau and in Poland; furthermore, along the marshy banks of great rivers, such as the Danube, Dnieper, Dniester, Don, and Volga, on the coasts of the Black and Caspian Seas, and on the great steppes.

In England malaria is encountered but rarely, and is endemic only on the eastern coast and in some of the lowlands of the Thames. Scotland and Ireland are free from it.

In France the disease is confined principally to the western and southern portions of the country. It extends eastward from the mouth of the Loire as far as Tours, and in a southerly direction along the entire western coast, which abounds in swamps and meadow-lands, almost to the Pyrenees. The southern coast, as far as the mouth of the Rhone, the plain at the junction of the Saone and the Rhone, including the city of Lyons, and the Department of Puy-le-Dôme, which abounds in swamps, are all prolific fever regions.

In Spain and Portugal, the most noted home of the malady is along the south-western coast, in the bottom lands of the rivers. Still it occurs, often in very malignant form, along the northern coast of Spain, in Galicia and Asturia, in the northern provinces of Portugal, and even on the uplands of Estremadura and Castile. It is also to be met with on the coasts, and in the marshy valleys and plains of Sardinia and Corsica.

Italy is, beyond question, the most malarious of all European countries. Aside from various humid regions in northern Italy, and that belt of country now belonging to France, including the cities of Milan, Mantua, Pavia, Nice, Venice, and Verona, the entire western coast constitutes a vast hotbed of malaria, which often extends eastwards as far as the foot of the mountains. This region begins at Leghorn, and extends through the Tuscan Maremma, the Campagna of Rome, the Pontine marshes, the malarious environs of Naples, and, with the exception of some mountainous regions, as far as the southern coast of Calabria. The entire eastern coast, on the contrary, suffers but little from the disease, the States of the Church being the only portion infested by malaria.

Fever is endemic, over large regions and often in a very malignant form, in the island of Sicily, the Ionian Islands, Greece and Turkey, including Bulgaria, Albania, Roumelia, Moldavia, Wallachia, and the vicinity of Constantinople.

Africa.—Along the western coast of Africa, in Senegambia, on the Guinea coast, and on the banks and islands of the Gambia, the Niger and the Senegal rivers, malarial fevers flourish to an extent and with a malignity scarcely equalled anywhere else. They also prevail, although in a less virulent form, on the eastern coast, through Mozambique and Zanzibar, as far south as Delagoa Bay; on the greater part of the island of Madagascar, and on the Comoro islands of Anjouan and Mohilla. They are furthermore to be found in southern Nubia, at the Upper Nile delta, at the junction of the two arms of the Nile, and especially on the banks of the White Nile; also in Egypt, particularly in Lower Egypt, occurring, here again, on the banks of the Nile, and in the moist regions of the delta; and also extending along the coast of the Mediterranean Sea. In Algiers malaria is very widely diffused, and very pernicious, not only on the coast, but on the oases, in the deep and damp valleys of the mountainous regions, and on the southern slope of the Atlas range.

It is said that the interior portions of Africa, of which but little is known, are also, in part, infested with fevers of a grave character.

America.—Certain of the islands of the West Indies rival even the western coast of Africa in the extent and the malignity of their fevers. To this number belong San Domingo, St. Christopher's, Jamaica, Dominica, and Tobago, all of which abound in humid valleys and marshy ground, while other islands, with more favorable conditions of soil, suffer little or not at all. In Mexico malarial diseases flourish on the eastern as well as on the western coast, whereas in Central America they are mostly confined to the western coast, from Cape Gracias-a-Dios to Panamá.

In North America these affections present themselves, often in very malignant form, in the Gulf States of Texas, Louisiana, Mississippi, Alabama, Georgia, and Florida; in the wide prairie lands between the Missouri River and the Rocky Mountains, and in the valleys of the middle and lower Mississippi and its tributaries. In the Middle States they are rare, and in the Northern States are probably unknown.

There is a dearth of reliable information with regard to the northern coast of South America, as well as concerning the southern portions thereof. We have accounts from Venezuela showing a considerable prevalence of the disorder. It is also to be found on the western coast of Ecuador and Peru, along the low banks of Brazilian streams, and here often in a malignant form, as well as in the interior of Brazil and Bolivia. Further, in the damp forest regions, heights and valleys of the Peruvian mountains and on the eastern slope of the Andes.

Asia.—A very extensive and malignant malarious region is to be found in the river districts of the Indus and the Ganges, which are annually overflowed by the water of these streams.

his is particularly the case with regard to the delta of the Ganes, and is also true of the shores of the Brahmaputra.

On the western coast of Hither India the disease is quite prevlent, while the eastern coast seems to be comparatively exempt. 'n the island of Ceylon, however, it prevails with a degree of irulence worthy of the West Indies or the Guinea coast. It is niversally prevalent in Farther India, as well as on the Sunda slands, especially in Sumatra, less so in Borneo, Java, and Celees; also on the Molucca and Philippine Islands.

In China the disease prevails along the entire southern and outh-western coast, and on the banks of the larger streams, with severity characteristic of the very worst malarious regions. It s endemic along the entire coast of Syria; along the northern oast of Asia Minor; in Arabia, on the shores of the Red Sea and he Persian Gulf; along the banks of the river Tigris; around he Caspian Sea in Persia, and most malignant on the elevated lateau of Teheran.

Australia.—Malaria may be found on the mainland of Ausralia, though in a mild form. It assumes a severer type on ome of the smaller islands, as the New Hebrides and the ociety Islands, and, strangely enough, is entirely unknown on he Sandwich Islands and the Samoan Islands, as well as the sland of New Zealand and Van Diemen's Land, in spite of their arshy ground and frequent inundations.

ETIOLOGY.

Malarial diseases are usually endemic in character, rarely poradic, and rarely advancing over large regions of country in he form of an epidemic.

Their endemic occurrence is especially common in marshy egions, and the more extensive these are, the more frequent and evere, as a rule, will be the diseases in question. But all arshes do not bear this relation to disease, and there are even xtensive swampy regions, in hot climates, that are entirely free rom malarial fever. For their influence varies with the amount f water they contain; where the latter stands high, fevers are ore rare; where the marshy ground is covered only by a thin sheet of water, and the latter is exposed to the heating influence of the sun, malarial diseases will abound, inasmuch as the decomposition of organic matter, and especially of vegetable matter, seems materially to aid in their production. Thus the most favorable conditions for the development of this poison are offered by marshes that have dried up, while their injurious influence is materially diminished as soon as heavy rains once more submerge the previously parched surface of the ground.

Marshes that are formed partly of salt water, especially delta lands, are far more noxious than those supplied entirely with fresh water. This is believed to be due to the action of the salt water, in killing the fresh-water plants, and thus producing a larger amount of decomposing material. It is difficult to determine whether or not other causes likewise contribute to this result, such as the salty ingredients of the soil, etc. Those marshes, furthermore, that rest upon a substratum of peat or sand are more wholesome than those resting on limestone, chalk, clay, or mud.

Instances occur, every now and then, in which, with every condition present for the development of malaria, this poison is entirely lacking. We cannot account for these exceptions unless it be on the ground of the disinfecting properties of ozone, which is said to be largely developed in some marshes. Examples of this sort are to be found in many of the islands of the Pacific, in the warm swamp-regions of the Australian coast, and, according to the recent accounts of Jourdanet,[1] in the city of Mexico, and its vicinity, a region offering all the conditions ordinarily resulting in malaria. Before the very gates of Mexico lies the lake of Tescudo, about twenty-five square miles in area, composed partly of fresh and partly of brackish water, with a clay bottom, which is often laid bare over large areas, as the result of evaporation, with a temperature of from 122° to 140° Fahr.;[2] and, notwithstanding all this, malarial fevers are rare.

The fact that marshes have a causative relation to malaria may be proved by the disappearance of the latter when marshes are drained dry and cultivated, and its reappearance when they are allowed to relapse into a state of nature.[1] This favorable effect of cultivation is partly due to the systematic removal of the surface-water, and partly to the absorption of decomposing organic matter by the growing crops. On the same principle, Maury succeeded in combating the malignant fevers that flourished in the marshes surrounding the observatory at Washington, by planting sun-flowers, which have an uncommonly great absorbing power. Sebastian recommends the culture of the calamus (acorus calamus aromaticus) for the same purpose.

---

[1] Union Médicale, 1862, No. 129.

[2] The original reads, "50°-60° Centigrade."—TRANSLATOR.

Systematic and thrifty culture of the soil, then, with its ditching and draining, and careful handling of all products, is the greatest enemy to malaria, which is found more rarely and in more benign form the more thickly inhabited is the land. On the other hand, it is very apt to be developed and to spread in an uninhabited region, where cultivation is neglected, where the ground remains untilled, and a luxuriant native vegetation is abandoned to its own destiny.

But it is not swamps alone that cause malaria. Damp bottom-lands, and regions that are exposed to an annual overflow, such as the deltas of rivers, with the heaps of mire thrown up on their banks, as well as lands with a clayey or alluvial soil, which presents an obstacle to the percolation of water ; all these produce the same results as swamps. In case of the working over or turning up of large portions of soil, as in the building of fortifications, of dykes, or of viaducts, in the rooting out of timber and the preparation of virgin land for cultivation—in all of which processes the animal and vegetable organisms hidden in the ground are brought to the surface, and rot under the influence of warmth and air; not only the laborers engaged in such enterprises, but the inhabitants of the surrounding country, far and wide, are liable to be attacked with the most violent malarial fevers. The "polders" of Holland, those portions of land reclaimed from the sea by the building of dykes, are notoriously of this character, and men working upon them are subject to the severest forms of the disease.

The sort of earthwork mentioned above may often give rise to the appearance of malaria at points where it had not previously prevailed at all, or to but a slight extent. As soon as the work is completed, the previous condition of health may be restored within the region concerned. Wenzel has made a report of the malarial diseases occurring in the Jade region during the twelve years occupied in the construction of a harbor, at the height of which epidemic one-half or two-thirds of the population were attacked during one month. The disease diminished gradually during the twelve years, without any other apparent cause than the changes taking place on the newly exposed surfaces of ground, whereby the giving off of the poison was progressively lessened. The fact that malaria may appear in places previously free from it, as soon as the conditions for its development exist, is confirmed by the unanimous testimony of Tessier, Blaxall, Lacaze, and others, concerning the epidemic that prevailed from 1866 to 1868 on the island of Mauritius, a spot previously exempt, and which had served as a health-retreat for those suffering from the fever in India. The heavy rains and inundations of one year had deposited great masses of vegetable detritus on the plains, which the unusually dry season of the succeeding year permitted to decay. In addition to this, great earthworks were built, rivers were cleaned out, forests were uprooted, and the mountain streams, spreading themselves out in the depths of the prostrate woods, created vast swamps. Finally, the crops failed, and great distress prevailed in the land.

---

[1] For instances of this, see *Hirsch*, Handbuch der historisch-geographischen Pathologie, p. 52, Note.

It has long been known that malaria may exist on a dry soil, or in mountainous regions, even at a considerable height, (although the dissemination of the poison is more in a horizontal than in a vertical direction), and that in the latter case it is often more extensive and severe than in the adjoining low country. On the Tuscan Apennines fevers are to be found at the height of 1,100 feet; on the Pyrenees, at 5,000 feet; on the island of Ceylon, at 6,500 feet, and in Peru, at 10,000, and even 11,000 feet. At the same time the neighboring plains are either entirely free, or are visited by the disease in a much milder form.

These apparent contradictions and exceptions to the rule are not always to be accounted for on the theory of the death and decay of organic matter. This much, however, is certain, that an elevated and apparently dry region, with a stratum of loose soil on the surface, and a deeper floor of clay or some other impermeable soil beneath, may present conditions favorable to the development of malaria, when a large amount of surface-water, loaded with vegetable ingredients, percolates through the loose upper earth, and is retained on the surface of the lower stratum. The best instances of this kind are found in the oases of the desert of Sahara. Hirsch describes these as consisting of trough-like depressions or excavations in a rocky or highly hygroscopic soil, the receptacle or river-bed of subterranean waters, which are covered with a layer of alluvium,—the surface of the oasis.

Here, and in other apparently dry regions, the intense heat of the sun often causes cracks and deep rifts in the earth, which give free exit to the miasm from beneath.

Whether similar conditions will be found to exist, explaining the origin of "mountain fever," such as a rocky soil, with clefts and chasms containing damp and decaying detritus, is not yet determined.

Instances are on record where earthquakes or volcanic eruptions have been followed by the appearance of malaria where it was previously unknown. It is difficult to account for such phenomena, all the explanations that have hitherto been given of them being purely conjectural. It is equally difficult to account for the prevalence and the disappearance of malarial fevers in certain places where no changes whatever have occurred in the relations of the soil; and we are thus forced to the conclusion that standing water and marshy soils are not the only factors concerned in the production of malaria.[1]

---

[1] According to the reports of Drs. *Luden* and *Corson* to the Pennsylvania State Med. Society (Canstatt's Jahresbericht, 1863, IV, p. 76), intermittent fevers prevailed along the banks of the Juniata river and the Pennsylvania Canal until the year 1856, when they suddenly ceased, without any known cause. In one year they appeared four miles higher up, on both sides of the river, and have never since been known below that point. In the same way, intermittent and remittent fevers were quite frequent in the Plymouth and Whitemarsh valleys until the year 1832, when cholera appeared; from that time to 1849 they grew milder and more rare, and now they have disappeared entirely, without any changes having occurred in the known conditions of the soil, woods, or anything else. *Böhm*, Eine Wechselfieber-Epidemie. Berl. med. Zeitung, 1860, No. 52.

The second important factor in the development of malaria is *heat.* The spread of intermittent fever is rare beyond the sixty-third degree of north and the fifty-seventh degree of south latitude; and from here to the equator the disease increases gradually in extent and intensity. Hirsch tried to determine the northern boundary of malarial fever more accurately, and found that the extreme northern points at which it prevailed in different parts of the world were at very various degrees of latitude and of average annual temperature. It thus became evident that in the production of malaria it was not the average annual temperature, but the average summer temperature that was of account, and that the northern limit of this, for fevers, lay between the isotheres of 59° to 59.8° Fahr. This is an elevation of temperature sufficient to insure the decomposition of vegetable organisms.

It is furthermore well known that the development of the disease usually takes place during the summer months, and that it disappears, or at least no new cases arise, during the winter, unless the latter is very mild. We know, too, that the form, type, and intensity of the fever are usually in pretty direct relation to the temperature of the atmosphere. This is not only established by the testimony of writers from warm climates, but instances are not rare in our own land of the influence of a hot summer in multiplying severe forms of the disease, and in raising the malarial poison to such a degree of intensity that it overleaps the limits of its usual endemic abode, and spreads abroad in epidemic form. No other instance of this need be given than the spread of remittent fever in the Netherlands during the hot summer of 1846.

The theory that *changes of temperature* conduce to the production of malaria, which is still advocated even by some writers of the present day, is abundantly disproved by facts. On the one hand, those countries where changes of temperature are most extreme, such as Scandinavia and the British islands, are comparatively free from malaria; on the other hand, those regions noted for an equable temperature are often the home of the most intense and pernicious forms of the disease.

Certain *climatic relations,* such as the season of the year, the amount of moisture in the atmosphere, and the winds, cannot be gnored in studying the genesis of malaria.

The season of the year is of consequence whenever it brings heavy rains, soaking the ground, which is subsequently exposed o the influence of a hot sun; and the period presenting such conditions occurs in different months at different localities. Although, as a general thing, this rule holds good, yet, according to Hirsch, great differences occur among different tropical egions, most of which have their fever season during and immediately after the rainy season. For instance, fevers are most prevalent in Senegambia and on the Guinea coast in June, Sepember, and October; in Zanzibar (Eastern Africa) from March o May, and from October to December; in the Bay of Delagoa northern limit of Caffreland) from the beginning of September o the end of April; in the West Indies during the entire summer and fall; in Central America from November to May; in Brazil from April to June. In subtropical regions, and in the more elevated and temperate portions of the tropical zone, fevers almost always arise during the summer, that is, at some period rom the end of June to the beginning of August, or even as late as September, and the maximum of their occurrence is from July o October. In colder climates (Germany, the Netherlands, Russia, Scandinavia, Great Britain, etc.), they most commonly appear in the spring, at the time of the snow's melting, often as arly as February; the maximum here is reached in May, a decided diminution of the disease being apparent in summer, folowed by a more serious outbreak in the fall, after harvest time. n the more temperate and colder climates the winters are usually free from the disease, though relapses and sequels of the disorder may then be met with, especially after repeated thaws have taken place.

According to the observations of Wenzel in the Jade region, a diagram of the annual prevalence of the disease [Erkrankungsurve] showed two elevations separated by a saddle-shaped depression, the elevations corresponding to the periods of spring and summer, the summer rise, during the several years, being usually higher than that of spring. The steep and rapid rise of he summer wave represents, in whole or in great part, new attacks, while its gradual and terrace-like descent in the autumn shows the diminution of new attacks, until it reaches the winter line of mere relapses, to be swelled again, although to a less height, by the new cases occurring in spring. The smallest number of new cases and of relapses took place during the summer, previous to the summer rise. It should be remembered that the greatest summer heat did not correspond, in time, with the greatest amount of sickness, but that the former preceded the latter by from twenty to twenty-five days, making it evident that about three weeks were required for the influence of temperature to make itself felt on the course of the disease. This period of time may be supposed to be divided between the development of the malarial poison and its incubation within the organism. Wenzel considers that about from six to eleven days should be assigned to the former period, and something like fourteen days to the latter, but it is evident that both periods must be greatly modified by circumstances. The various degrees in which the conditions requisite for the production of malaria (organic matter, heat, and moisture) are present must modify the former; and the quantity of the poison brought to bear in any given case, as well as the susceptibility of the individual attacked, must influence the latter.

It is hardly to be doubted that the degree of moisture of the atmosphere and the amount of rain-fall have some influence on fevers. In the tropics the latter are found to be more extensive and more severe the more damp the year has been, and comparatively infrequent and mild after a prolonged dry season. Even with us, malaria is evidently fostered by a wet spring and summer, followed by a hot autumn, or by a wet spring and a hot summer followed by a wet autumn, or by a very rainy year succeeded by a very dry one. Jilek has demonstrated, by the following table, that in Pola, a noted malarious district of Istria, the extent of the epidemic corresponded with the amount of rain that fell:

Year.	Fall of Rain.	Number of persons in every 100 attacked with fever.
1864	18.44″	51.4
1863	14.25″	48.6

Year.	Fall of Rain.	Number of persons in every 100 attacked with fever.
1866	12.10″	36.3
1865	8.44″	35.4
1867	5.49″	22.9
1868	1.5″	14.2

A high degree of atmospheric moisture, in a malarious district, according to Bechi, produces an indescribable unrest, lassitude, and muscular debility even in strong, robust men, leading to attacks of fever in more susceptible individuals. This effect of moisture and of evaporation shows itself, in the most marked degree, during the first few hours after a severe shower following a prolonged dry season.

This is true not only of dry fever regions (Griesinger), for I have observed the same thing in damp Holland, during a dry summer and autumn. I cannot determine whether Boussingault's[1] observations, to the effect that rain-water contains much more ammonia after a long, dry season than after continued rain, and that this is taken up by the atmosphere after rain, the more quickly the warmer the air is, and the more the condition of the soil favors evaporation, have any bearing on this subject or not. Griesinger believes that they warrant our attributing to ammonia a special influence in the production of fevers.

Lancisi was among the earlier writers who recognized the agency of the wind in aiding the spread of marsh fevers, by virtue of its power of carrying material disease-germs. He attributes to the influence of the winds the fact that the Roman Campagna became more unwholesome after the removal of the sacred groves, and its consequent greater exposure to the miasm of the Pontine marshes. Similar testimony may be found in all ages, and of the most varied kind.[2]

---

[1] Academie des Sciences, 1853. Session of the 28th of November. Griesinger, p. 9.

[2] *Gesscle* (Bair. ärtzl. Intelligenzblatt, 1867, No. 43) reports that only one case of intermittent fever occurred among forty to fifty laborers, engaged for two years in cutting turf, or peat, on the northern shore of the Chiem-see, some of which laborers

In later years, Barat accounts in the same way for the epidemic that arose in 1869, on the island of Reunion, believing that it was borne over by the wind from Mauritius. From the 7th of April to the 23d of July, 4,118 people, out of a population of 23,000, were attacked with the disease, and no other cause could be found for it, seeing that the state of the soil, and all other conditions that could exert any influence in its production, had remained unchanged. However plausible such reports may sound, they do not always sufficiently account for the spread of malaria. Hirsch very properly calls attention to the fact that cities and large stretches of country often lie between the original fountain of poison and the point where the disease has broken out, and that these, although apparently more exposed to the malarial influence on account of their proximity, are often entirely exempt. We may admit the agency of the wind as a carrier of miasm for a short distance; but when it comes to stretches of many miles, it is no longer to be taken into account.

On the other hand, it is well known that in malarious districts the disease increases materially during hot, dry, still months that have been preceded by moisture, and that it is often greatly diminished as soon as fresh, strong currents of air cleanse the atmosphere.

It is claimed that the wind from certain directions,[1] the east wind in England and the sirocco in Italy, have an influence favorable to the development of fevers. According to Hirsch, Salvagnoli and several other observers are of the opinion that malarial diseases increase in extent and intensity, in Italy and

---

had come from non-malarial regions. On the other hand, a very extensive epidemic of the fever broke out in the village of Truchtlaching, about an hour distant, which was itself entirely free from marsh or moorland. Gessele accounts for this fact by the strong breezes that blew over the peat-bog, and which reached the village, which lay in a valley to the south-west, open only on the side toward the contagion.

[1] *Moffat* (Assoc. Med. Journ., Aug. 30th, 1856) found, as the result of meteorological observations, extending over a period of five years, at Hawarden, that the widest spread of the disease was coincident with a fall both of the barometer and of the thermometer, and with the prevalence of southerly and westerly winds, including all those points of the compass from south-east to south, and from south to north-west; also, that the maximum mortality was coincident with a falling of the barometer, and with prevailing northerly and easterly winds, ranging from north-west to north, and from north to south-east.

Sicily, on the appearance of the sirocco; that these diseases then penetrate further inland, and that at such times even strong, healthy men lose flesh and appetite, and suffer great bodily and mental prostration. Hirsch believes that the influence of the sirocco, and probably also of other winds that are supposed to have some special relation to malaria, depends on thermo-atmospheric conditions or agencies, that is, on changes in the degrees of heat and moisture present.

The fact that, during the endemic prevalence of malaria, only a certain number of persons are attacked by it, the majority remaining exempt, although subject to the same telluric and climatic influences, forces us to search for individual predisposing causes, which may account for this difference. Some writers, such as Schwalbe,[1] believe that the reception of the malarial poison into the system is not enough to produce disease, but that some vice of constitution must exist, in addition, in order to destroy the equipoise of the organism and call into activity the latent poison. In evidence of this, those cases especially are adduced in which persons who have lived in a malarious region for some time, free from the disease, develop the same after removing to a distant and entirely non-malarious district.

In view of all this, let us consider, briefly, the influence of the following conditions.

No race or nationality enjoys immunity from malarial affections, and if it has been determined that the negroes are less liable than others to be attacked by them, both in their own homes and in other malarious regions, it must be because they possess greater powers of resistance to the noxious influences which predispose to malarial diseases, or, in other words, because they have become inured to them.

All periods of life, from infancy to old age, are liable to attacks of malaria. But the forms of disease seem to vary somewhat according to the age of the subject. Children under five are said to suffer most frequently with intermittent bowel-troubles.[2] During the period of youth, either continued fevers or pure intermittents, quotidian or tertian, are most common. In middle life, when most of the cases occur, all forms are to be met with ; while old men, though less liable to infection, when once attacked, often suffer from the disease in its most pernicious form.

---

[1] *Schwalbe*, Einige Bemerkungen über Malariafieber. Archiv f. Heilkunde, 1867, Heft 6, p. 567.

[2] *Wunderlich*, Handbuch der Pathologie und Therapie. Stuttgart, 1854. Bd. II, Abth. 1, p. 562.

Men are more subject to malarial affections than women, which may be due to their greater exposure to the chances of infection. A certain degree of immunity has also been claimed for pregnant and puerperal women. Thus Quadrat[1] claims that during an epidemic at Prague he saw only two cases of intermittent fever among 8,639 pregnant and puerperal women. The observations of other reporters, however, are at variance with this. According to Ritter, the most that can be claimed is an apparent immunity on the part of pregnant women, who are less liable to contract the disease during the last months of pregnancy.

The statistics of the Lying-in Hospital at this place would also argue against the existence of any such immunity.[2] Malarial attacks are not rare during childbed or while nursing the infant, although under these circumstances they are most likely to be relapses of a disease existing before labor. Some writers maintain (Ritter) that during the puerperal period the rhythm of the attacks is irregular and the apyrexia not well defined.

Women are more likely to be the subjects of a masked intermittent, especially showing itself as neuralgia in the branches of the trigeminus nerve. Men, on the other hand, are more liable to the severer forms of pernicious and remittent fever, at least this is true in Holland. This is doubtless because the men, from the nature of their work, laboring in the soil and the "polders," are exposed for a long time to the influence of the most concentrated poison.

---

[1] Oesterreich. med. Wochenschrift, 1841, July 31.

[2] I owe the collection and analysis of all cases at this institution to the kindness of my friend and colleague, Prof. Lehmann, who has also communicated to me his experience in this matter during the more than twenty years of his active service within its walls. During the years 1850 to 1871, 8,686 cases of labor are on record, with 182 attacks of intermittent fever in different individuals. This is about $2\frac{1}{10}$ per cent.; but it must be remembered that very light cases were omitted from the account. With a business of about 400 labors annually, the lowest proportion of malarial attacks was, for 1862 and 1869, each year two, or one-half of one per cent.; the highest ratio was for 1857, which showed thirty cases to 332 pregnant women—nearly 10 per cent. Forty-five of the 182 noted above ($2\frac{4}{10}$ per cent.) had relapses during the puerperal period.

Varieties of constitution are not to be ignored, as influencing the liability to the disease. Weak and anæmic persons are the ones most liable to be attacked. According to Griesinger, marked differences in the character of the disease may sometimes be recognized, according as it occurs in subjects of a plethoric or of an anæmic constitution.

All weakening influences must be reckoned among the specially predisposing causes: such are hunger, thirst, loss of sleep, various bodily and mental exertions, emotional disturbances, fright, anger, exhaustion, and fatigue, and all such unfavorable incidental occurrences as sea-sickness, menstruation,[1] etc. Increased moisture of the atmosphere, from any cause, has the same effect, such, for instance, as may result from the frequent washing of floors, especially in bedrooms, sleeping in damp beds, excursions by water, promenades at morning and evening during fogs, sitting out-doors after nightfall, especially during damp weather, when the ground is wet after rains, or in the vicinity of bodies of water, whether they be standing or flowing. Residence in cellars and on ground floors, or anywhere in the immediate vicinity of the soil, is injurious, especially if such apartments are used as sleeping-rooms; the same is true of the immediate proximity of high trees to dwelling-houses, inasmuch as they prevent the free ingress of sunshine, keep the ground damp and cold, and prevent the purification of the atmosphere.

An outbreak of the disease is sometimes brought about in the inhabitants of valleys where intermittent fever prevails, by their removal to a cool mountain region. In fact, every sort of cooling down, dry as well as moist, especially if the body has been particularly heated, may give rise to the development of malarial affections. The same is true of excessive solar heat, of indigestion, of inordinate eating and drinking, and of the imbibing of stale drinks or of swamp-water.

---

[1] *Sebastian*, loc. cit., p. 48, says, that women are usually attacked by the epidemic fevers at the time of menstruation.

The action of many of the injurious agencies mentioned above, as regards their influence in the development of malarial disease, must be explained by their disturbing the equilibrium of the body, increasing debility, and thus diminishing the power of resistance to the malarial poison. Whether moisture of the atmosphere, and remaining in the open air after sundown, have any other effect than what is exercised by their cooling down the body; whether, as some assert, the malarial poison exists in more condensed form in the midst of fog and dew, and at a low temperature; or whether the absence of the sun's rays modifies the chemistry of the marsh miasm, are questions that are at present hard to decide.[1]

Undoubtedly previous paroxysms of intermittent fever constitute the greatest predisposing cause for renewed attacks of the same; whence we must conclude that, after the first invasion, some changes unknown to us are wrought in the organism, which are capable, without any intermediate manifestations, of leading to a new outbreak of the disease on the slightest provocation, and which gradually disappear if no new infection takes place.

We may thus speak of a certain latency, or period of incubation of the poison, which exists, for a greater or less period, also among new-comers in a malarious region, before the first attack of true intermittent fever occurs. Sometimes this latent condition of the poison persists until the individual has again removed from the fever region. Such individuals, as well as those who have suffered from the fever in a malarious region, but have been free from it for some months, may, after a while, be attacked by the disease again, while in a part of the country entirely free from fevers, or even while living in high mountain regions.[2]

This doctrine of the latency of the poison accounts for relapses, on the supposition that they are merely a new development and outbreak of the disease-germs that have been slumbering within the system and are now awakened to life. This wakening may depend on various causes, such as "taking old," a fit of indigestion, changes of temperature, etc. Per-ons who have but newly arrived in a malarious region resist hese influences less successfully than those who are acclimated, nd in the tropics this is particularly true of light-haired, blue-yed Northerners, while negroes and those of mongrel breed ndure the climate better.[1] Strangers are more liable to suffer oth from simple intermittent and from remittent and perni-ious fevers, while natives present rather a state of chronic ickliness, or succumb to the acuter forms of fever only during ore than ordinarily active epidemics, and even then are not so iolently attacked. It appears, therefore, that there is no such hing as absolute acclimatization, which would enable the or-anism to offer successful and continued resistance to the spe-ific influences of the climate, but only a comparative power of esistance against the less virulent poison. The degree of accli-atization which is attained after a prolonged sojourn in a alarious region amounts merely to an accommodation of the ystem to the prevailing evil influences, and this is accomplished y the foreigner's adapting himself, as early and as completely s possible, to the habits of the country, as regards diet and ress.

---

[1] *Plagge*, Deutsche Klinik, 1858, No. 1.

[2] I have demonstrated the truth of this in my own person. Although, during the first years of my residence in Holland, I had suffered much from malarial disease, at one time having an eight-days' attack of continued fever, yet I had been free from it for several months when I took up my abode in a part of Germany that was entirely free from malaria, and in which no case of intermittent fever had ever occurred. After about four weeks I was seized with an intermittent attack, which yielded to quinine, but which recurred some weeks later, though in a milder form.

Malaria sometimes breaks loose from its endemic haunts and hows itself in places where it has seldom or never before ppeared. It thus loses its endemic character and sweeps over onsiderable regions of country as an epidemic, or over vast sec-ions of the globe as a pandemic.

In the present state of our knowledge, we have no thorough nsight into the conditions that govern the periodical rise of pidemics. If we take into account the observations that have een made in sundry smaller, more circumscribed epidemics that ave occurred in some fever-lands, we shall find that they point to ertain atmospheric conditions as a probable cause. In several murderous epidemics that ravaged Holland during the seventeenth and eighteenth centuries,[1] uncommonly great and prolonged heat existed, as well as unusual dryness of the air and absence of wind, so that the waters dried up, and exhalation from the swamps was immensely increased. In other countries, too, a great increase of moisture, with very hot seasons following it, has been the signal for epidemic outbreaks of the disease. Among familiar instances of this are the epidemics of fever that occurred after the enormous floods on the north-west coast of Germany in 1717, which were followed by two years of great heat and dryness; and after the vast overflow of the German Ocean and the Baltic in 1825, which was also followed by two hot summers.

---

[1] *von Frantzius*, Virchow's Archiv, Bd. 43, p. 328.

In view of what has been said before, it does not seem probable that currents of air are capable of carrying the poison which is generated in the breeding-places of epidemics to a distance of any considerable number of miles. We believe rather that malarial poison is, in the majority of cases, generated on the spot; inasmuch as the conditions necessary for the decomposition of vegetable matter may be furnished in a non-malarious locality by alterations in the conditions of the soil, by overflows, great solar heat, etc. It need hardly be said that we do not claim thus to fathom all the causes that contribute to this end. The annual variations which are found to exist in the frequency and the severity of endemic outbreaks may also be attributed to similar telluric and atmospheric influences.

It is a still more difficult matter to account for those isolated areas of malarial poison which are often confined to a single street, or to one side of a street, or even to single houses, unless, indeed, we attribute them to the above-mentioned telluric influences, supposing them to arise from subterranean swamps and collections of water, the exhalations from which reach the surface through rifts in the ground. The sporadic cases of intermittent fever found occasionally in non-malarious regions may undoubtedly often be attributed to previous infection in some fever district. It may also be remarked here that some paroxysms of chills and fever may occur which are not intermittent in character, but are more of a nervous nature, attacking persons of a very sensitive organism, or which may depend upon some unrecognized organic disease.

---

[1] The epidemic in 1669 is described by *Sylvius de Le Boë*, Praxeos medicæ app. Tract. X, § 63, 65, 67. *Guidon François*, Dissert. medica de morbo epidemic. Leidæ, 1671, and *Koker*, De morbo epidemico, 1719.

Malarial epidemics and pandemics, of which a considerable number have shown themselves during recent centuries, seem to stand in some thus far inexplicable relation to epidemics of other diseases, especially to those of typhus fever, cholera, plague, dysentery, and influenza, which have either preceded or immediately followed the march of malaria. The first epidemic of this character, which spread over the whole of Europe, took place in the year 1558; it was preceded by a widespread influenza in 1557, and followed by the ravages of plague from 1559 to 1563. The second malarial pandemic, which was followed by an extensive visitation of the plague, lasting for three years, occurred in 1678 and 1679. The third appeared during the years 1718 to 1722, and was succeeded by a general outbreak of typhoid fever. After some smaller epidemics which occurred during the last century in Germany, Holland, and France, but not simultaneously in all of these countries, the fourth pandemic flourished from 1807 to 1812, again following influenza, spreading over nearly the whole earth, and being itself followed by an epidemic of typhoid fever. The great malarial epidemic before the last was in 1824 to 1827, and the last in 1845 to 1848, each of them being the forerunner of another scourge, viz., the typhoid epidemic that prevailed from 1826 to 1831, and the cholera which invaded Europe at the beginning of the third and the end of the fourth decade of this century.

A certain interchange between malaria and the diseases mentioned above may also be traced in some of the smaller epidemics. Thus influenza and malaria seldom prevail simultaneously, although they often follow and would almost seem to exclude one another. On the other hand, intermittent fever and dysentery often occur side by side, both endemically and epidemically, the former even being complicated by the latter, without our necessarily supposing that there is any relationship between the two miasms. Epidemics and endemics of intermittent and typhus fever are not rarely to be met with at the same time, and in the course of my professional life in Holland I frequently observed that, during the extensive prevalence of intermittents, cases of typhus fever, some of which are always to be found there, became more numerous. The same thing does not hold true with regard to typhoid fever, the epidemic appearance of which is not often simultaneous with that of intermittent fever. I do not assert that there is a law of complete mutual exclusion, as to time and place, between these two diseases, but this much I can declare, from my own experience, that in Amsterdam, where all forms of malarial fever are indigenous, typhoid fever is among the greatest of rarities. The annual statistics of 2,000 cases of sickness, in the division of medical diseases, show barely one or two cases of typhoid fever.[1] An entirely different relation exists between cholera and malaria; for we find certain regions, as in India, where the two flourish side by side. In the cholera epidemics of 1831 and 1848, on the other hand, intermittent fever disappeared, to break out again on the cessation of the cholera,[2] or to remain suppressed for years, in some places where it had previously been endemic.

The question of the immunity from tuberculosis due to the influence of malaria, which has been claimed and fought over by various writers (Wells,[3] Nasse, Heusinger, Boudin,[4] *et al.*), has assumed an entirely new aspect under the light of recent investigation. (Buffalini,[5] Ritter.[6]) The most striking evidence in this matter is furnished in Holland, as is shown by the reports of Dutch and Belgian physicians (Nieuwenhuis,[1] Schneevoogt,[2] Grooshans,[3] Schedel[4] and Gouzee[5]), as well as by the latest observations of Masse[6] in Algiers. According to my own experience, tuberculosis and the phthisis resulting from cheesy pneumonia are very frequent diseases in the fever districts of Holland, and especially in Amsterdam, affecting not only the lower classes but also those in good circumstances. These pulmonary affections are often associated with repeated attacks of intermittent fever, followed by the malarial cachexia. The fact that intermittent fever also occurs later in the history of these affections (as was observed ninety-nine times in 381 phthisical patients by Schneevoogt), is not difficult of demonstration, if we regard the heavy chills and fever which regularly recur at a given day and hour, usually assuming the tertian or quotidian type.[7] I must disagree with the statement of Nieuwenhuis, that the course of phthisis in Holland is not so rapid as in southern Germany; if intermittent fever is grafted on to the pulmonary affection, the progress of the latter seems to be accelerated. The many conflicting views that obtain with regard to the existence or non-existence of phthisis and tuberculosis in malarial regions show us something of how we should regard the question of an antagonism between these diseases. The non-existence of one or another disease is to be accounted for, not by the presence of intermittent fever, but by certain characteristics of the climate. According to Duchek,[8]

---

[1] Similar reports are made by *Stiefensand* (loc. cit.), with regard to the rarity of typhoid fever in the malarial regions of the Rhine; by *Spengler* (Casper's Wochenschrift, 1848, No. 21), with regard to Rheingau, where typhoid fever increased on the diminution of the malarial fevers which had previously been endemic; and by *Volz* (Haeser's Archiv, IV, p. 531), concerning the rarity of typhoid fever in malarious regions.

[2] *Wunderlich* (Handbuch der Pathologie und Therapie, II, 1 Abth., p. 591) has been able to demonstrate this by the varying use of quinine before, during, and after the cholera epidemics of 1831, 1837, and 1848.

[3] Transactions of Society. London, 1872, III, p. 417. *Horn*, Archiv, 1818, Bd. 2, p. 330.

[4] Traité de fièvre intermit. 1842.

[5] Gaz. Toscan., 1847.

[6] *Ritter* claims (Virchow's Archiv, Bd. 41, p. 239) that miliary tuberculosis exists alongside of intermittent fever in all malarious regions. On the other hand, he asserts that cheesy pneumonia, which derives its origin from solid foreign bodies suspended in

---

the air, which irritate the lungs mechanically and chemically, necessarily does not exist in malarious regions, as malaria is developed in the presence of abundant moisture and of decomposing vegetable organisms, which are unfavorable to the development of pulmonary tuberculosis.

[1] Topographie méd. d'Amsterdam. 1817.

[2] Gaz. méd., 1845, No. 32.

[3] Verslag over de inwendige Klinik, etc. Utrecht, 1847.

[4] Gaz. méd., 1845, No. 32.

[5] Arch. de Belge, 1842, Juill.

[6] Rec. de mém. de méd. milit., 1868, Févr., p. 124. Compare also the bibliography of Griesinger's Infectionskht., p. 13, note.

[7] *Ward*, Lectures on Intermittent Fever, Lancet, 1864, Oct. 16th, and 29th, saw sailors, in the marine hospital under his charge, suffering at the same time from malarial fever and tuberculosis.

[8] Wiener Spitalszeitung, 1859.

epidemics of intermittent fever are often preceded or followed by the prevalence of bronchial catarrh, while pneumonia, rheumatism, and scrofula are very frequent in malarial regions.

In case of the extensive endemic spread of intermittent fever, or during a true epidemic of the same, other diseases catch its typical feature, that is, the symptoms belonging to them assume a pretty well-marked remission and exacerbation, which can easily be controlled by quinine. Intermittent fever may also simulate other diseases, inflammatory and non-inflammatory; it even sometimes localizes itself in single organs, as will be shown more clearly under the head of symptomatology.

The different forms of malarial disease differ from one another only in degree, being all dependent on one and the same poison, subject to variations in quality and quantity. They may be arranged in a progressive scale, from the lightest to the most severe, beginning with the quartan intermittent, passing on to the tertian, the masked intermittent, and the double tertian, and thence to the remittent, the continued, and the pernicious fevers; the different grades bearing a pretty direct ratio to the intensity of the poison.

As a rule, the grades of disease last mentioned belong to the tropical and subtropical regions, while the first, or lighter forms, exist in temperate and cooler climates. But this rule is by no means without exceptions. Although the quartan intermittent is one of the greatest of rarities in tropical climates, yet the simple tertian occurs often enough; and again, severe and pernicious forms of the disease may be developed in temperate and cool climates. From all of which it would appear that this matter is determined not by degrees of latitude, but by telluric and atmospheric influences, as well as individual tendencies. Unfavorable conditions of the ground, moisture, and great heat, favor the production of malarial poison in the highest degree. When, therefore, these conditions arise in temperate climates, as has been the case in a number of epidemics, we must not be surprised to meet with the most severe tropical forms of fever. On the other hand, a cool season, with favorable conditions of the ground, resulting in the production of but a small amount of poison, may lead to a mild form of the disease, even in the tropics.

The concentration of the poison, then, determines the severity of the disease. During the twelve years' epidemic in the Jade district, according to Wenzel, very hot summers were accompanied by severe forms of the malady, such as remittent and pernicious fevers. The warmer months were characterized by those attacks which approached the continued type, viz., quotidian intermittents; the cooler months presented those types which show a longer interval, viz., the tertian and quartan. In studying the malarial fevers of the Caucasus, Papoff[1] found the quartan type to prevail from November to January, the tertian early in the spring, passing, towards the end of that season into the quotidian, and giving way to the remittent and continued forms during the summer; in autumn the tertian type reappeared, to be followed, in turn, once more by the quartan. The same changes in type may be observed in individual epidemics, the height of which, accompanied by the severer forms, is usually reached during the summer, while their decadence, characterized by milder attacks, takes place in the cooler months. For, during the hot season, the concentration and intensity of the poison are heightened by the far more rapid decomposition of organic substances, by more active exhalation, and by the greater capacity of the atmosphere for taking up these exhalations.

The intensity and rapidity with which the morbid processes in the body are developed depend on the more or less powerful as well as the more or less continuous influence or working of the poison. It is hardly necessary to remark that these conditions, as well as the very form of the disease itself, are greatly influenced by the peculiarities of the patient, the fact of his having previously had malarial fever, his powers of resistance to specific injurious influences, general debility, antecedent illness, organic disease, mental or bodily fatigue, unfavorable habits of living, etc.

It appears that during the course of pernicious fever the participation of certain organs in the processes of disease depends partly on individual peculiarities (that is, the diminished powers of resistance of one or another organ), and partly on the temperature and the time of year. During the summer-time, when the temperature is high, we may observe a marked tendency to implication of the nervous system and the digestive organs, showing itself in diarrhœa, dysentery, choleraic and bilious attacks, as well as in typhoid forms, while during the winter the tendency is more to catarrhal and inflammatory affections of the respiratory apparatus.

---

[1] Med. Zeitung Russlands, 1857, No. 32 et seq.

Up to the present time but little is known of the nature of the malarial poison. The older observers (Mascati, Vaucquelin, Fontenelle) merely demonstrated the presence of decomposing organic matter in marsh-exhalations, and the theory has long been generally accepted that malarial poison is exclusively the result, in a gaseous form, of the decomposition of vegetable organisms (such as carbonic acid gas, carburetted hydrogen, and, according to Schwalbe, carbonic oxysulphide).

In opposition to this view stands the fact that some marshy regions which present all the conditions for the development of such products of decomposition (some districts in Alabama, Peru, and elsewhere), some of which are even surrounded by the most notorious malarious regions, remain exempt from the disease. It is no rare occurrence, where malaria prevails, to find that the sort of weather which, according to this theory, should favor its production (moisture, a high temperature, etc.) is accompanied, not by an increase, but by a diminution of the disease. Furthermore, a number of extensive malarious districts are known which are entirely free from the influences attributed to marsh-lands, from surface-water, dampness, etc. Among this number, according to Hirsch,[1] may be reckoned the high plateaus of Castile, the plains of the Araxes, the terrace lands of Persia, the Tuscan marshes, the Roman Campagna, Calabria, the table-lands of India, the island of Kutch, and the island of Ceylon.

The necessary conclusion from all this is, that the telluric and atmospheric influences referred to above are *not* sufficient to account for the origin of malaria, and that there must be additional causes at work, thus far unknown to us. Some think that they find these causes in the exhalations from living plants, in the ethereal oils,[1] inasmuch as the prevalence of the disease corresponds, not with the decay, but with the highest development of the plants. Others attribute the poison to subterranean exhalations, to the gaseous effluvia from a volcanic soil; still others deny any specific cause, or, again, believe it to consist in an accumulation (Eisenmann) or modification of the electricity of the earth and the air.[2] None of these views have as yet been established by sufficient proof; and even the supposed microscopic vegetable organisms, of low grade, the spores and algæ,[3] with regard to the influence of which in producing malaria so much has been said, lack, as yet, all practical confirmation.

---

[1] *Hirsch*, Handbuch, I. p. 54.

Another question, on which opinions are very much divided, is, in what way the poison enters the organism, and what organ is first and prominently attacked by it. It appears as if the most natural way for the reception of the poison was through the respiratory apparatus, and yet some observations indicate that the same thing may take place through the intestinal tract.[1]

---

[1] *Vaughan*, Philadelphia Med. and Surg. Reporter, Dec. 16th, 1871.

[2] According to Armand (Traité de climatologie générale, Paris, 1873), the thermo- and electro-hygrometric phenomena of the atmosphere, by their combined action, their intensity and changeability, constitute the remote causes of fever; while the immediate causes lie in the changes produced in the entire organism, and especially in the nervous system, under these influences.

[3] This theory has been brought forward again, of late, by various observers. *Thomas* (Archiv f. Heilkunde, VII, p. 225). *Scoda* (Clinique Européenne, 1859. Canst. Jahresber., 1859, IV, p. 73) believes them to be either living or in a state of decomposition. *Baxa* (Wien. med. Wochenschr., 1866, p. 78) saw low, cell-like structures in drinking-water. *Balestra* (Compt. rend., LXXI, No. 3, p. 235) discovered a species of algæ in the Pontine marshes. *Salisbury* (Amer. Journ. of Med. Sc., 1866, Jan., p. 51) found in his investigations in the valleys of the Ohio and Mississippi, that the sputa of the sick contained small elongated cells, presenting themselves singly or in rows, which he considered to be alga-cells of the species palmella. These he also found and collected on glass plates set up over marshy ground, and in great quantities on the clods of an upturned marshy soil. According to his observation, these alga-cells do not rise over 100′ above the level of the sea. He was able to produce the most intense attacks of intermittent fever by means of the fresh clods, if allowed to place them within the open window of a sleeping-room in a house lying about 300′ high. The attacks, in four persons, the subjects of two experiments, followed in ten, twelve, and fourteen days, and were broken up by quinine. *Hannon* (Journ. de Méd. de Bruxelles, 1866, Mai, p. 497) says that when he was devoting himself to the study of the sweet-water algæ, during their fructification, he was attacked with an intermittent fever of six weeks' duration. In opposition to Salisbury, *Harkness* states (Boston Med. and Surg. Journal, 1869, Jan. 14th) that he has found the palmella spores in the snow and at the summit of the highest Alps, and claims that they may very readily become mixed with the saliva and the urine from without, at the same time having nothing at all to do with malaria.

The disease has been observed to arise from the use of foul drinking-water; and yet it is a question worthy of consideration, whether the use of such water has been the direct and immediate cause, or whether, with its injurious, but not specific ingredients, it has only served as an occasion for the outbreak of the disease, in a person already infected with malaria, a view which has, of late, been especially advocated by Colin, and with which my own experience leads me to coincide.

The doctrine of the communicability of intermittent fever from man to man, as is taught by some, has little in its favor, and much against it. The direct demonstration of this, with the exclusion of all other possibilities of infection, would be a difficult matter.[2]

Some believe in the communicability of malarial fever through the mother or wet-nurse to an infant at the breast. Boxa[3] declares, that in Pola ninety per cent. of the children suckled by mothers or nurses with malarial fever, were attacked with the disease.

---

[1] *Merieu de Saint Gilles* (Influence des miasmes marécageuses sur l'économie animale. Montpellier, 1829, April 9, und Canstatt's Jahresbericht, 1865, IV, p. 65) produced trembling and stupor in three young squirrels by the administration of two spoonfuls of the "dew" or condensed vapor collected from over a pond. His father drank half a glass of this fluid, which immediately produced nausea and cardialgia. In subsequent experiments, in which the swallowing of the water was immeately followed by the use of quinine and opium, no such effects were produced.

Boudin (Traité de fièvres intermit. Paris, 1842, p. 66) says, that of 120 soldiers who returned from Bona to Marseilles on the ship Argo, in the year 1834, 103 were attacked with the most various forms of malarial fever, some of them pernicious, because during the voyage they drank of the marsh-water that was taken on board at Bona. On the other hand, the sailors of the same ship, who had good water, and 780 men who were embarked on two other vessels, remained well. Those soldiers on the Argo who were not attacked bought their drinking-water of the sailors.

[2] *Büchner* (Aerztliches Intelligenzblatt, 1860, No. 26) reports, that a man travelling from a malarious region slept with another man, and the latter was seized with malarial fever.

[3] Wiener med. Wochenschrift, 1865, No. 42.

The tendency of domestic animals, in malarious regions, to be attacked with the disease is much less than that of men. In Holland, East Friesland, and Westphalia, there seems to be nothing of this, but more of it is found in southerly latitudes. Common intermittent fever, usually of the tertian type, has been observed in horses, cows, dogs, and hogs; pernicious forms have also been seen (Rivolta mentions nine in cattle), as well as malarial cachexia, with a tumor of the spleen, and spontaneous rupture of the tumor.

PATHOLOGY.

Careful observation appears to show that the period of time which intervenes between the introduction of the malarial poison, or the infection of the system, and the outbreak of the disease, in other words, the *period of incubation*, may be of variable length. It is commonly reckoned at from six to twenty days, that is, an average of fourteen days ; but exceptions to this rule are by no means rare. I have repeatedly satisfied myself that the disease may appear immediately after the reception of the injurious influence. For this purpose I have sat down beside marshy ditches which were in process of drying, selecting those in particular that were distinguished by their putrid exhalations. As soon as within half an hour I would experience a slight disturbance of vision, ringing in the ears, dizziness, a feeling of roughness and burning in the throat, nausea, choking sensations, and chilliness, and a few hours later an attack of fever, which was usually light.

Salisbury states that when, in the pursuit of his investigations, he passed over a stretch of marshy moorland, almost bare of water, the soil of which was thrown up by the cattle that grazed there, he used to experience a peculiar sensation of heat and dryness in the fauces and throat, extending down to the bronchi, and accompanied by a constant impulse to hawk or clear his throat. On the other hand, the period of incubation may very easily exceed, by a good deal, the time mentioned above as being normal; some trustworthy observations to this effect having been made quite recently. Blaxall reports four cases among the crew of a man-of-war that had spent five days in the harbor of Port Louis. Two of these were attacked, with the quotidian type, in twelve and fourteen days, two others, with the tertian type, at the end, respectively, of forty-eight and one hundred and eighty-four days after embarkation. Pfeiffer[1] states, from his own observation, that among the Weimar troops which were quartered near Flensburg, in August and September of 1848, besides the occurrence of a few cases of the disease on the way home, an extensive outbreak of intermittent fever took place after an interval of nearly six months. He also shows the same state of things to have existed among the troops that were quartered in the casemates of Rastatt in 1866, and who did not succumb to the disease until April, May, and June of the following year. Similar cases are mentioned by Braune.[2] He was able to obtain information with regard to the subsequent history of twelve of the bathers who were on the island of Borkum, belonging to East Friesland, in the autumn of 1868. Eleven of these had intermittent fever, two suffering with it before leaving the island, and afterwards having relapses, the remainder not being attacked until the following spring and summer, at their homes, in wholesome, and, for the most part, elevated localities. To this may be added the communication of Fiedler[3] concerning a lady in Dresden who developed the disease nine months after being at Borkum; and concerning five other persons, who had been at the sea-baths of Norderney, and who developed malarial fever after a period of latency of one, six, and ten months.

General Course of the Disease. Simple Intermittent.

An attack of simple, benign intermittent fever is not seldom preceded, in persons inhabiting a malarious region, by a prodromal stage lasting from six to eight days, or even longer, which in itself shows but little that is characteristic, and justifies no conclusions as to the disease which is to follow. The symptoms consist in general disturbances, such as occur also in other ute infectious diseases, and often suggest typhoid rather an intermittent fever. They may show various modifications. he symptoms are often so insignificant that the patient is subcted to no disturbance in his business, consisting of short tacks of frontal headache, darting pain in the eyes, slight itching of both eyelids, diminished appetite, chilly, creeping nsations over the body, a disposition to stretch and yawn, and nally, profuse sweating at night. Sometimes the first manifestions occur in a previously diseased organ; thus, persons with eak digestion suffer first from pyrosis, with fulness and presre in the epigastrium, giving rise to the suspicion perhaps of a lapse of their previous difficulty. Generally, however, the proromal symptoms are much more severe. They consist of lasside, a feeling of exhaustion, and unwillingness to move about uch, which is usually followed by copious sweats. Combined ith this is a feeling of depression and mental confusion, a disste for mental or physical effort, frontal headache, dizziness, ague, wandering pains, shooting pains in the limbs, especially the calf of the leg, and pain in the back. Sleep is uneasy, ften disturbed, and accompanied by distressing dreams. The atient feels a sensation of fulness and tension in the epigasrium, is obliged to draw deep inspirations, complains of nausea, nd often actually vomits. The taste is stale, bitter, often metalc, rarely acid; the mouth and gums are dry, the tongue and eth often covered with a slimy coating, and the former white round the edges and yellow in the centre. At the same time here is loss of appetite, repugnance to animal food, a foul reath, constipation or diarrhœa, and considerable thirst. The atient is very sensitive to changes of temperature; sensations of eat and cold alternate in the body; the skin is generally hot nd dry, the complexion pale and muddy, growing yellowish and lightly icteric about the sixth to the eighth day. The pulse is gular, though small and frequent. The urine is scanty, darkolored, and turbid, containing an abundance of urea, and often ausing a burning sensation on micturition. Even as early as his, the spleen is usually a little tender on pressure, or during a eep inspiration, and somewhat swollen. The foregoing pheomena usually assume remissions and exacerbations in such manner as to become developed into true characteristic paroxysms of fever, with chill, fever, and sweat in due form, these paroxysms gradually becoming divided from one another by more distinct intervals of apyrexia. Or, when these symptoms have been maintained at about a uniform grade for several days, a violent chill may intervene, marking the beginning of the actual attack.

---

[1] Jena'sche Zeitschr., Bd. IV, Heft 1.

[2] Archiv f. Heilkunde, 1870, XI, Jahrg., p. 68.

[3] Archiv f. Heilkunde, 1870, p. 425.

In other instances the prodromal stage may be entirely lacking, and the disease may begin with a pronounced chill, which is subsequently repeated at regular intervals. This is especially liable to be the case with strangers, who have moved from a region where malaria is unknown into one where it prevails, or with persons who are attacked in a non-malarious region.

The actual *paroxysm of fever* is characterized by three stages, which are generally easy to distinguish. The first, the *stage of chill*, begins by the patient feeling uncomfortable, weary, worn out, and sleepy, often yawning, stretching, and feeling like lying down. In addition to this, he suffers from headache, dizziness, dragging pains in the neck, back, loins, and extremities, as well as a subjective feeling of cold; a sensation of chilliness running over the surface of the body, usually beginning in the back or loins, sometimes in the extremities, and spreading in every direction, this being at first relieved by intervals of warmth. These gradually disappear, and the chill, which is very variable in intensity, becomes constant; in the lighter cases it may merely amount to a hardly recognizable shudder, in the graver ones there is trembling of the lips, chattering of the teeth, twitching of the extremities, and a convulsive shuddering of the entire body; at the same time the pain in the head, back, and extremities increases in intensity.

The skin is dry, pale, and shrunken; the muscles of the skin are contracted, producing the peculiar condition known as goose-flesh (cutis anserina). The face is pale, the eyes sunken, the nose pointed, the lips and nails blue, the fingers white and cold, and the skin on them wrinkled and numb, as if dead, from deficient arterial circulation. The mouth is dry, the voice weak and indistinct, and, by reason of the trembling of the lips, broken and tremulous. There is often nausea, retching, and vomiting of recently ingested food. Patients complain of anxiety, palpitation of the heart, constriction of the chest, and thirst. The pulse is small and frequent, hard, and often irregular. Respiration is short and rapid, often anxious and sighing; the eyes are dull and tearful, the pupils dilated. Fainting, delirium, or stupor are very rare in simple intermittent fever; when they occur, it is in very irritable subjects. The urine is usually increased in quantity, clear, and watery, of low specific gravity, and without sediment. The temperature of the remote portions of the body is below the normal standard; in the mouth, armpit, and rectum it is above.

The duration of the stage of chill is quite variable, ranging from a scarcely noticeable period to from a quarter of an hour to one or two hours, and may even last for from four to six hours.

The *second*, or *hot stage*, also develops itself gradually. Trembling of the limbs becomes less, or, for a time, ceases altogether, to be renewed now and then, either spontaneously or from slightly uncovering the body, moving the bed-clothes, etc. Between times, sensations of heat are experienced; at first slight, afterwards stronger, which travel from the extremities to the trunk, and here, according to the statements of the patients, feel as if they were working from within outwards. At last this warmth permanently replaces the feeling of cold. The trunk becomes burning hot, the temperature of the periphery is elevated, and the appearance of the patient changes. His face becomes flushed, his eyes red and fiery, the tip of his nose—for a long time stone-cold—grows warm, the livid color of the hands and feet disappears, the skin of the body becomes smooth again, its natural turgescence returning, and the volume of the limbs is increased. Herpetic vesicles sometimes appear about the mouth. Pain in the back, loins, and extremities is relieved, while the upper portion of the abdomen and the regions of the spleen and kidneys are often painful, either spontaneously or on pressure. The head is in a glow, the headache increases, the impulse of the heart is augmented, the carotids throb, the pulse rises in frequency, and is full and hard. Respiration is accelerated and noisy, but the feeling of oppression is relieved. The patient complains of a roaring in his ears, and of seeing sparks; he is very restless and much excited, a condition which, in children or very irritable persons, may increase to delirium. The lips, tongue, and mouth are dry and hot, there is urgent thirst, and the pupils are often dilated. The urine, which is voided but sparingly, is red, and of high specific gravity.

This stage usually lasts from three to four hours, sometimes only one or two, or even less, and, in severe cases, six, eight, or twelve hours.

After a longer or shorter time the third, or *sweating stage*, begins, with a diminution of the burning heat. The skin in the axilla and groin, on the forehead and the breast, becomes gradually moister, and finally the entire body is covered, at first with a gentle perspiration, afterwards with a copious, steaming, sour-smelling, and sometimes sticky sweat. The skin at the same time grows paler, the tongue and mouth moist, and the thirst less annoying. The patient's intellect is undisturbed; he feels relieved, as to his head; the unbearable pains diminish, and eventually disappear; the breathing becomes freer and easier, and the general condition of the patient is so improved that, during this stage, he often drops into a quiet sleep. The pulse becomes softer, wave-like, and regular, and diminished in frequency. The temperature rapidly sinks to the normal point. Urine is passed freely; it is of high specific gravity, rich in solid ingredients, and throws down a brick-dust sediment of the urates.

The duration of this stage is not easily determined, as the transition to a state of apyrexia is usually gradual, and the patient feels tolerably well even during the sweating stage. As a rule, this stage materially exceeds either of the others in length.

The entire paroxysm, with its three stages, generally lasts from four to twelve hours, more rarely from eighteen to twenty-four, or even longer, thirty to thirty-six. The latter scarcely occurs in the ordinary fevers, but only in the malignant quartan type, with scarcely recognizable periods of apyrexia.

With the diminution in sweating the patient passes into a state of apyrexia, and commonly falls into a quiet sleep. On awaking, in the lighter attacks and in short paroxysms, he usually feels quite well, except a little weakness and weariness, and can ordinarily pursue his avocations with no great difficulty. Often, however, especially in cases of more prolonged attacks, the head is a good deal disturbed; dizziness and certain gastric disturbances remind the individual of the ordeal he has just passed through. The tongue is then coated, the taste metallic, the appetite diminished, the epigastrium slightly distended and full, the bowels irregular, and the skin a little yellow; there is also marked sensitiveness to changes of temperature. The patient feels tired and exhausted, complains of pains in his limbs, ringing in his ears, and dimness of vision; he is very irritable, and on moving about or making the least physical effort he breaks into a sweat. The regions of the spleen and liver are, as a rule, somewhat sensitive on pressure; the urine is sometimes dark, and throws down a brick-dust deposit. Now and then the pulse may become frequent and the nights disturbed. Such imperfect intermissions, with a rapid pulse, occur after the first attack of fever, and may be repeated in case the disease is prolonged. In this way an intermittent fever may pass into one which is only remittent.

Certain deviations from the course above indicated may occur, but they are of such a nature that while they modify the features of the disease they do not alter its essential character. There are cases, usually of the quotidian or tertian type, in which, at a given hour of the day, a slight feeling of chilliness is experienced, followed immediately by heat, glowing cheeks, dizziness, headache, and redness of the eyes, lasting for several hours, and not succeeded by a sweat; or the chill lasts for a few minutes and the patient thereupon begins to sweat profusely; or, chill and heat may both be absent, and the fever paroxysm betray itself only by a periodical sweat, which usually occurs at night, and during which the thermometer will indicate a rise of temperature of from one to two degrees. Such instances are not rare among people living in a malarious region, who are but slightly influenced by the poison, either from the latter being of low intensity or from the individual possessing unusual powers of resistance; they also occur as a species of relapse during recovery from fever.

The intensity of the several stages may differ widely in the same individual in successive paroxysms, the stage which was longest in one paroxysm being, perhaps, shortest in the next. Even in severe cases of intermittent fever, some stages may be very short or entirely lacking, and this has usually been regarded as an unfavorable sign. The disease may begin with the period of heat, the missing chill being replaced by some severe brain symptom, as delirium, sopor, or convulsions.[1] Saint-Vel[2] reports instances, in the intermittent fevers of Saint-Pierre, Martinique, in which the chill was entirely absent, or consisted of a slight shudder, and the hot stage lasted from twelve to thirty-six hours, often accompanied by the gravest symptoms, and followed by but slight sweating. It is more rare to find an intermittent fever, of any considerable gravity, presenting the chill and heat without sweat, and still more so to find the sweating stage alone present; at least, none of the latter cases have come under my own observation. It is claimed that intervals of several hours sometimes occur between two stages of a paroxysm, as between chill and heat, or between heat and sweat (febris dissecta). According to some isolated reports there may be a reversal of the order in which the stages follow one another, as, for instance, where the chill follows the heat and sweat, or where the attack begins with heat, followed by a chill and ending in a cold sweat.[3] Griesinger,[4] it is true, believes that most of these are cases of quotidian fever, in which the night sweat of one paroxysm ceases shortly before the morning chill of the next. When successive paroxysms follow one another so rapidly that the chill of the second occurs during the sweating stage of the first, we designate it as febris subintrans, and it constitutes a transition stage to the remittent and subcontinued type of fevers, in which only remissions and exacerbations, or only slight, hardly perceptible fluctuations occur.

---

[1] *Graves*, Clin. lec. soc. ad 1848, I, p. 357. *Mohl*, Vereinszeitung, 1856, No. 4. *Günsburg*, Günsburg's Zeitschrift, Bd. VII, Th. 5.

[2] Gazette hebdom., 1863, No. 13.

[3] *Canstatt*, Prager Vierteljahrschrift, 1850, p. 92.

[4] Infectionskrankheiten. *Virchow*, Specielle Pathologie und Therapie. Bd. II, Abth. 2, Ed. I, p. 19; II, p. 23.

The older physicians were perfectly familiar with the paroxysms, recurring at regular intervals, which constitute the essential characteristic of intermittent fever. We divide the fever into *types* corresponding to the length of these intervals. For the attacks recur pretty regularly every twenty-four hours (intermittens quotidiana), or every forty-eight hours (int. tertiana), or every seventy-two hours (int. quartana).

The type which occurs most frequently, in temperate climates, is the tertian; it is the one in which the paroxysms are most clearly defined and the intermission (apyrexia) usually complete.

With us the quotidian, with its imperfect intermissions, is more rare, though it is a form under which the disease not infrequently begins. This, as well as all short types approaching the continued form, is more commonly met with in the tropics, or in our climate during a hot summer. The quartan type is still more rare, and is said not to exist at all in the tropics. It is more likely to show itself in the course of a prolonged attack, and is accompanied by certain irregularities in the course of the different stages, as well as by an imperfect intermission, due to the existence of the malarial cachexia.

Those types of fever which present a still longer interval between the paroxysms are even greater rarities; in fact, their existence is denied by many. Such are the fevers which occur every fifth,[1] sixth, seventh,[2] eighth,[3] or even every thirtieth day.[4] In addition to the simple types of fever, *double types* may also occur. Two paroxysms may appear daily, at different times of day, and of different intensity (int. quotidiana duplicata); or one attack may appear daily, but of such form that the paroxysms of the first and third, and those of the second and fourth days correspond with one another as to time of day and intensity, presenting not the quotidian type of fever, but two tertian combined (int. tertiana duplicata). Some claim to have seen als a double quartan, in which two fevers, recurring on the fourt day, and distinguishable from one another by the time and t intensity of their duration and symptoms, occurred in the sa individual on different days, being so arranged that the first a fourth day belonged to one fever, and the second and fifth the other. This brings two successive fever days together a leaves every third day free. To these may be added one mo form, which does not occur with us, but which, according Sprengel,[1] prevails occasionally as an epidemic in Italy, Hu gary, the West Indies, and Bengal, and which was alluded by the oldest writers, although not correctly interpreted by the It is the semitertian intermittent (the ἡμιτριταῖος), which consis of a quotidian and a tertian combined, so that on the first and t third days there are two attacks each, while on the second d there is but one.

---

[1] Referred to, among older writers, by *Marcellus Donatus*, *Tulpius*, *Joh. Matth. Müller*, *Werlhoff*, *Stoll*, and *Kurt Sprengel.*

[2] *Thilenius*, Nassauer Jahrbücher, 1863, Heft 19 and 20, p. 221.

[3] *Binz*, Deutsche Klinik, 1867, No. 24, observed an octana in a boy six years old.

[4] *Valerius*, of Taranta, described the thirty-day form as early as the end of the fourteenth century. *Saint-Vel*, Gaz. heb., 1863, No. 13, after seeing quintan, sextan, septan, and octan types, finally also found one recurring on the thirtieth day.

The double types seldom appear as such primarily, but ari in the course of a prolonged attack, or appear in the form relapses.

It often happens that the paroxysms, while maintaining th rhythm, recur a few hours too soon (*anticipating*)—and this the more usual form—or too late (*postponing*).

If the anticipating or postponing process is frequent repeated, it does alter the rhythm of the fever. Thus, by anti pating, a quotidian is changed into a remittent or a subco tinuous form, a tertian into a quotidian, and a quartan into tertian; the reverse of all this being accomplished by postp ing.

In other and rarer instances, especially among persons w have suffered repeatedly from malarial disease, it is liable show the greatest irregularity and variety in the occurrence a character of the paroxysms (intermittens erratica). It is oft observed that after apparent recovery from intermittent fe a relapse will take place at the end of a week or a month, t patient having in the meantime felt well; and this relapse w fall on a day which would have been a regular fever-day had the original attack continued its rhythm. This holds true with regard to the tertian type, the paroxysms recurring on odd days, and still more with regard to the quartan type (Graves, Griesinger), which often suddenly reappears on the regular day, after an interval of a month.

---

[1] Handbuch der Pathologie. Leipzig, 1807. II Band, p. 156.

The attacks of tertian fever usually take place towards noon, those of the quotidian variety in the morning or at noon. As a rule, they fall into the hours between midnight and noon, much more seldom into those between noon and midnight. Paroxysms beginning in the afternoon and evening hours are rare,[1] and were considered by the older writers as of ill omen, Peter Frank believing that they were indicative of a hectic origin.

In children intermittent fever shows some deviations from its ordinary course. As a general rule, children suffer from the quotidian type much more frequently than from the tertian or quartan. The differences worthy of mention are seen in children in the first two years of life, after that there is but little difference between children and adults. The disease may appear in infants two months old. At the beginning of the attack the child becomes quiet and sleepy, the skin grows cool, pale, and wrinkled, the lips and nails become blue, the nose pointed, and the eyes sunken; at the same time the pulse is small, often scarcely to be counted, and the respiration short and frequent. The trembling and shaking, which is so well marked in adults and older children, is altogether absent. After a period of from ten minutes to an hour, the hot stage follows. The patient becomes restless and excited, the face grows red and glowing, the pulse stronger and fuller (120 to 130), the carotids and fontanelles pulsate strongly, and the skin is dry and hot, as are also the lips and tongue. Sometimes children fall into a stupor, or older ones may be seized with convulsions. After an hour to an hour and a half the pulse falls, and a slight, generally partial, perspiration breaks out, this never being so general and profuse as in older children and adults. Aside from these fully developed paroxysms, it is not uncommon to find incomplete forms in which the chill is lacking, and the hot stage with a very light sweat constitutes the attack. The intermissions are rarely complete. Children generally remain sleepy, irritable, and morose, with the appetite poor, and the functions of the bowels disturbed.

---

[1] *Mohl* (loc. cit.) observed them in the epidemic of intermittent fever which prevailed in Nicolai in the months from July to September, 1855.

The most severe cases may run their course under the guise of eclampsia, with extreme pallor and coldness of the skin, unconsciousness, dilated pupils, convulsions, etc., ending in death.

It is claimed that cases of *intra-urine malarial disease* have been observed[1] in which the children were born with œdema of the feet and an enlarged spleen; it is said that they slept much and cried but little, that they were alternately cold and hot, pale and red, that they afterwards suffered again with œdema of the face, hands, and feet, and showed all the evidences of the malarial cachexia, which, however, yielded to quinine, though not without occasional relapses.

Masked Fevers.

Masked malarial fevers—the name is not happily chosen—are those attacks of disease which, bearing the type of an ordinary intermittent, present symptoms foreign to this malady; which ordinarily run their course without fever, or are accompanied by partial febrile manifestations, as slight chilliness or heat, with a rapid pulse or sweating; and which depend on an affection of certain nerve-tracts.

These masked fevers usually assume the quotidian type, more rarely the tertian or quartan; still, other types may occur, and it is not uncommon to find remissions in the place of intermissions. They appear both by day and by night, usually during the morning hours, and are, as a rule, of about two hours' duration.

These fevers either appear as independent diseases, in persons otherwise well, or they are associated with other forms of sickness, or they may precede or follow ordinary intermittent fever, or alternate with the latter in a regular manner, so, for instance, that the first day shall present a regular attack of fever, the second the masked form, the third again the regular paroxysm, and so on, assuming the character of a double tertian. The spleen is seldom enlarged, and the urine does not often throw down a sediment.[1] Masked intermittents usually show themselves as *typical neuralgias*, in the course of one or another nerve trunk, not to be distinguished by any means from other neuralgias. The character of the pain is very various. It may break forth suddenly in its fullest violence, or it may, for several days, give warnings of its approach and gradually increase in intensity. According to Griesinger, masked malarial attacks are most frequent in persons over forty years of age, only exceptionally occurring in those under twenty.

---

[1] *Basin*, Gaz. des hôp., 1871, No. 72.

The most common seat of these neuralgias is in the course of the trigeminus nerve, especially in the frontal branch of the ophthalmic division. They are liable to appear daily, during the morning hours, and are often associated with throbbing pain in that side of the forehead, swelling of the upper lid, injection of the conjunctiva, and running at the nose, as well as nausea, retching and vomiting, or, it may be, with general malaise, chilliness, heat, and sweating, and an elevation of temperature demonstrable by the thermometer. The earlier attacks usually last but a short time, hardly half an hour, the later ones, which are very likely also to be anticipating in character, continue for two, three, or four hours. More rarely the neuralgia will be found located in the superior or inferior maxillary division of the trigeminus, in the occipital nerve, or in the intercostal nerves. Typical neuralgia of the sciatic nerve, a not infrequent affection, is either intermittent or continued, though, in the latter case, having decided remissions; it is sometimes accompanied by tonic or clonic spasms, is usually one-sided, occurring more frequently on the right side than on the left, seldom on both sides. Schramm,[2] in thirty-four cases of this affection, found it to exist twenty-two times on the right side, nine times on the left, and three times on both sides. The instances in which this disease is found located in the crural, the peroneal, the popliteal, or the tibial nerves, in the mammary gland or the thoracic organs, are more rare. When occurring in the latter region it usually consists in attacks of pain, more or less severe, about the region of the heart, shooting down the left arm, accompanied with violent palpitation, anxiety, a cramp-like sense of constriction, stertorous breathing, cold skin, pale or livid countenance and lips, fainting, and loss of consciousness. Ward[1] reports a typical case of præcordial neuralgia, or angina, which lasted for several hours, and Bierbaum,[2] a similar one of neuralgia of the vagus nerve, which began with yawning and stretching of the limbs, and was followed by dyspnœa, cough, aphonia, and dizziness. Neuralgia of the tongue occurs but seldom; cardialgia, on the contrary, is common, especially in Holland, and is often ushered in by a slight chill. It sometimes begins with a feeling of unpleasant oppression, which gradually increases until it amounts to the most severe pain, frequently accompanied by a scarcely perceptible pulse, cold extremities, great anxiety, eructations, vomiting, and thirst. These attacks are usually of the anticipating quotidian type, sometimes tertian, and occasionally, in the gravest cases, remittent; they are as likely to appear in the evening or night as during the morning. Typical colic, with obstruction or diarrhœa, is of very uncommon occurrence, and the same is true of neuralgia of the pharynx, the scrotum, the urethra (with occasional retention of urine), and of the uterus.

---

[1] *Griesinger*, loc. cit., edit. II, p. 47, discovered albumen in the urine in a case of supra-orbital neuralgia.

[2] Aerztl. Intelligenzblatt, 1859, No. 34.

Anæsthesia[3] along the course of various nerves is a condition arising much less frequently than neuralgia. Among the ills of this nature seldom met with are typical cramps, attacks of sneezing, coughing (sometimes with sibilant rhonchi), hiccough, eructations, tremors, and convulsions in single limbs, clonic and tonic spasms in the course of the facial nerve, and hysteriform and chorea-like seizures. It is claimed that typical cramp of the vessels supplying certain extremities has been observed.[1] Now and then intermittent paralysis of certain limbs is met with, hemiplegia, paraplegia, or paralysis of the tongue and organs of deglutition.[2]

---

[1] Lancet, Oct., 1864.

[2] Deutsche Klinik, 1862.

[3] *Schramm*, loc. cit., saw a crural neuralgia, of seven days' standing, disappear under the use of leeches, but during the following night it appeared in the sciatic nerve of the same side, with the greatest violence, and the outer surface of the thigh presented a condition of complete anæsthesia (anæsthesia dolorosa typica).

We may, furthermore, meet with cases of typical mutism, aphonia, aphasia, macropia and micropia,[3] amblyopia, amaurosis,[4] night-blindness, and deafness; as well as certain psychical disturbances, delirium with hallucinations, maniacal attacks, especially during the puerperal state, and typical vigilance (Trousseau), in which the patients, without any special evidences of sickness, spend every second or third night, according to the type, entirely without sleep.

Masked fevers may, furthermore, manifest themselves in the form of intermittent hyperæmia, hemorrhage, œdema, and inflammatory disturbances of nutrition. Among the latter are typical coryza, bronchial catarrh, swelling of the tonsils, the tongue,[5] and the liver, the latter being accompanied with severe intermittent pain; various skin affections, as erysipelas, purpura, roseola, urticaria, and pemphigus; hemorrhages from the nose, the lungs, the kidneys, the uterus, the intestines, and the stomach; œdema of the lower extremities or of the mamma, ascites, and anasarca; furthermore, typical iritis and ophthalmia (which latter, if prolonged, may lead to atrophy of the globe), usually introduced by fever, severe pain, photophobia, and contraction of the pupil.

Finally, we must mention certain still more rare disturbances, such as typical attacks of severe thirst, typical diarrhœa, intermittent vomiting of the contents of the stomach and small intestines (typical merycismus),[1] expulsion of gas by the mouth or by the anus,[2] which is often introduced by a slight chill and dizziness. Intermittent jaundice also is sometimes encountered. During the year 1859, Martin[3] saw a whole epidemic of intermittent jaundice, of the tertian type, among the French garrison of Pavia. On the fever days, the spleen and liver being swollen, the discomfort was greater, and the urine, conjunctiva, and skin were darker colored than on the days that were fever-free, and the disease was rapidly subdued by quinine.

---

[1] *Raynaud*, Canstatt's Jahresb., 1862, IV, p. 69.

[2] *Macario* (Gaz. méd. de Paris, 1857, No. 6) reports an interesting case of this kind, in which a woman was seized, two days after her confinement, without any known cause, with formication in the feet, which then spread to the thighs, the trunk, and the upper extremities, these becoming, at the same time, paralyzed and anæsthetized, and her tongue also becoming paralyzed. These manifestations were repeated three times, after the quotidian type, and were then arrested by quinine.

[3] *Retsin*, Moniteur des Hôp., 1859, No. 29. Canstatt. Jahresb., 1859, IV., p. 85.

[4] *Richard*, Gaz. des Hôp., 1863, No. 107.

[5] *Riccardi* (Moniteur des Hôp., 1857, No. 21. Canst., 1857, IV, p. 177) describes a case of glossitis, in which severe pain in the tongue arose during the chill, this organ becoming greatly swollen during the fever, which swelling subsided after the sweat. These appearances were shown again during a second attack, and quinine prevented a third.

It was stated above, that masked fevers might alternate with regular intermittents, according to some definite type; in the same way the different forms or localizations of masked fever may alternate with one another, or with the pernicious variety of the disease. I once had under treatment a lady who was suffering with quotidian supra-orbital neuralgia, and who, when this was relieved, was attacked with sciatica of the same type. Borelli[4] reports a similar case. A woman suffered from periodical spasm of the neck of the bladder, of the double tertian type; after this had been cured by a few large doses of quinine, and she had felt perfectly well for five or six days, she was seized with all the symptoms of cholera, and upon recovery from that developed typical sciatica.

Pernicious Fevers.

Malarial fever may become dangerous to life when it attacks individuals who, on account of their constitution or their age, are unable to offer sufficient resistance to its assaults; or, because the symptoms belonging to the disease are developed with especial violence, and the individual stages are of too prolonged and exhausting duration; or, again, because the disease has localized itself, in a threatening manner, in some important organ.

---

[1] *Clemens*, Deutsche Klinik, 1857, No. 51.

[2] I met with a case of this kind about a year ago, in the person of a man some thirty-five years of age, who suffered daily, at a given hour, with general discomfort and chilliness, accompanied by the most prodigious escape of wind by the anus, lasting for several hours, a condition which was cured by the use of quinine.

[3] Moniteur des sc. méd., 1861, No 144.

[4] Canstatt's Jahresber., 1860, IV, p. 74.

Simple, uncomplicated malarial fever may end in death, in the case of children, very old people, or those who have been reduced by previous disease, when the intervals between the febrile exacerbations are short, the fever temperature is high, and medical aid is not summoned in good time. In weakly and irritable children eclampsia is very liable to supervene, the little patients lying unconscious, with dilated and sluggishly reacting pupils, frequent pulse, and elevated temperature, while the muscles of the face, the neck, and the extremities are subject to violent convulsive twitchings. Such paroxysms may last for several hours—as long as six—and be repeated, with greater violence, on the next day or the third day. With timely help, the milder forms may recover, but paresis or paralysis frequently remains as the result.

In aged people, and those otherwise enfeebled, the disease sometimes becomes dangerous by exhausting the last remaining powers of life, rapidly assuming the adynamic character. But occasionally the duration and intensity of the individual paroxysms or stages may be so considerable as to threaten life by the collapse thereby induced, growing weakness and irregularity of the pulse finally terminating in paralysis of the heart.

The extreme hyperæmia and swelling of the spleen, common during the chill and the hot stage, may lead to apoplexy of the spleen, to rupture and the escape of blood into the peritoneum, or to gangrene of the organ. Some years ago I treated a woman in my clinic who had for a long time suffered with the most violent intermittent fever. At the autopsy of her case, we found the spleen covered with a greatly thickened capsule, and the organ itself the seat of an extravasation of blood half as large as a man's fist, which had led to suppuration, perforation, and fatal peritonitis.

Those fevers which are benign in form, and only terminate unfavorably on account of peculiar circumstances, often dependent on the individual attacked, are sometimes reckoned as pernicious fevers, although *pernicious fevers, in the proper sense* *of the term*, are only such as are characterized by special, dangerous, local affections in important organs (febres intermittentes perniciosæ, seu comitatæ). These local affections, which usually assume the intermittent type, but which may also appear irregularly and entirely mask the malarial fever, are liable to simulate a good many other diseases, and thus to obscure the diagnosis.

It is held by some that under such circumstances we have to deal with two distinct diseases, viz., an organic disease and an accidentally coincident intermittent fever, or *vice versâ*. There is, however, no doubt that the former is developed under the influence of the malaria poison, and is to be considered merely as a peculiar localization of the disease, one of the best arguments in support of which lies in the favorable results of quinine treatment.

Pernicious malarial fever not infrequently appears in the form of an epidemic, though sporadic cases of it may be found in the somewhat more intense epidemics of ordinary intermittent. In some malarious regions, as in Holland and in Hungary, as well as in the warmer climates of Italy, northern Africa, and the tropics, especially in the East and West Indies, and western Africa, numerous instances of the disease may be observed, without any epidemic influence.

The *prodromata* of pernicious fever, when there are any, are generally not to be distinguished from those of simple intermittent, and it is but seldom that the dangerous paroxysms are preceded by special symptoms, such as severe headache, dizziness, great drowsiness, convulsive twitchings, or grave intestinal disturbances. The pernicious symptoms rarely accompany the first attack, usually appearing with the second or third paroxysm, or after several paroxysms of a simple character have supervened. Thus, in the quotidian type the pernicious attack is usually after the second to the fifth days; in the tertian type, often later yet, sometimes not till the end of two or three weeks. The simple forms either gradually pass into the pernicious by a progressive aggravation of their symptoms; or a simple attack, of not unusual length and severity, may suddenly be followed by a very pernicious one, this seldom proving fatal the first time, but only on being repeated for the second or third time.

The *paroxysms* of pernicious fever are usually of the same duration as those of a simple character; sometimes, however, they are longer, lasting for twenty-four hours or more. The separate stages of chill, heat, and sweating may be more or less well marked, or they may be lacking in part or in whole, and may be followed by complete or incomplete intermissions, which are often very brief and hardly perceptible. They sometimes assume the quotidian, at other times the tertian type, very seldom the quartan, or one having a longer interval.

Pernicious fever occasionally passes back into the simple intermittent form again; when treated early it is also liable to show evidences of the former affection on the regular fever-days.

The form and the local affections of pernicious fever appear sometimes to depend on external or individual causes. During the hot season of the year disturbances of the nervous system and of the digestive apparatus are most common, during the cooler season, those of the respiratory organs. Present or previous disease of certain organs, or special influences brought to bear upon them, may determine the localization of the symptoms. I have seen this produced, as regards the brain, by injuries of the skull, through a fall or a blow, or after unusual exposure of the head to the sun's rays, or after emotional disturbances, or finally, after the excessive use of spirituous drinks. An epidemic influence seems sometimes to be an element in the matter, inasmuch as all the fevers of a given epidemic present the same or similar characteristics.

In general, the following forms of pernicious intermittent may be distinguished.

The one most frequently observed is accompanied with severe symptoms of *disturbance of the nervous system*, producing prolonged attacks, very irregular in their course, and terminating in a tedious convalescence. One of the most common forms is the comatose, which has also been designated as intermittens apoplectica. This seldom shows itself in persons after simple intermittent fever without complications; as a rule, the last ordinary attack and the intermission following it show some not unimportant signs of impending ill. Excessive headache and dizziness are liable to exist, as well as disturbances of speech, apathy, and extreme drowsiness, which tend to increase. The expression of countenance, at the same time, becomes stupid, and the features relaxed; answers are given slowly, briefly, imperfectly, and finally not at all. After the chilly stage, which is sometimes accompanied by convulsions, the patient, during the fever which ensues, falls into a state of sopor, unconsciousness, and coma. He lies there with a hot, flushed face; eyes closed; pupils wide and immovable; the skin hot, dry, and often yellowish; the tongue and mouth also dry, and often covered with a fuliginous coat; the breathing rapid and stertorous; the pulse sometimes fast and sometimes slow, full or small; the body generally insensible, and the limbs fully relaxed. The patient may remain in this condition for ten, twelve, or twenty-four hours, until he comes to himself in the midst of a profuse sweat, weary, worn out, confused, and complaining of headache and dizziness, all of which disappear after a while under appropriate treatment. Sometimes a certain impediment in speech remains for a time, or even permanently—a stuttering, a certain obtuseness—paresis in some one of the limbs, more rarely muscular contraction; or the comatose condition may last without change for several days, the heart's power sink more and more, the pulse grow weaker and irregular, and the patient die, at last, in such an attack. Even in case of a favorable issue, the prospect becomes worse with each successive paroxysm, and aid should therefore be furnished promptly. The disease may also assume a different form, the patient, during the cold stage, falling into a succession of fainting fits lasting for about a quarter of an hour, and accompanied by a small, frequent pulse (febris intermittens syncopalis). Sometimes the first attack of this nature proves fatal, but the patient may recover from it, and, after a short stage of heat and of sweating, may fall into a refreshing sleep, free from fever. Usually, even if the first attack does terminate favorably, the patient succumbs to one of the succeeding ones.

Under this form of pernicious intermittent must also be classed those cases of *apparent death*, which may last from half an hour to four hours. Persons subject to such attacks may remain entirely conscious, seeing and hearing everything that occurs or is said around them, but unable to move or to utter a sound; or they may be entirely unconscious, respiration arrested, pulse and heart-beat not to be recognized, and even the sharpest irritants applied to the body calling forth no signs of life until, at the beginning of the sweating stage, the patient comes to himself, and the various organs again slowly manifest their activity. Trousseau reports the case of a man who had had fainting fits on two occasions in Algiers, and in a subsequent attack fell into this condition of simulated death. It was not until he had been carried into the post-mortem room that evidences of life were observed about him, whereupon he was returned to his bed, and recovered under quinine treatment.

A still more uncommon form of pernicious intermittent is that which shows great *cerebral disturbance with delirium.* This form, too, seldom appears entirely unannounced, but is introduced by severe headache with dizziness, seething and roaring in the head, rushing and ringing sounds in the ears, restlessness and vigilance. The delirium varies from that of the lightest grade to the most violent maniacal attacks, with wild screams, curses, and blows, and hallucinations of sight, hearing, and touch. The face is flushed, hot, and streaming with perspiration, or, in reduced and anæmic persons, pale and sunken. The eye is glazed, the pupil dilated, the carotids beat violently, and the skin of the entire body is hot and dry. This condition lasts, at the outside, for a few hours. The patient may die suddenly of collapse during the attack, or, on the gradual lowering of the excitement, may fall into a deep coma from which he never wakes. In favorable cases the delirium becomes gradually milder, profuse perspiration follows, and the patient falls into a prolonged sleep, from which he awakes quiet, though still very weary, with headache and dizziness, but without the slightest consciousness of what has taken place. In this form of the disease, too, a repetition of the attacks is in the highest degree dangerous.

Severe nervous symptoms, in pernicious intermittent, may show themselves in other ways, too, giving evidence, apparently, of a simultaneous affection of the brain and spinal cord. Such are clonic and tonic forms of spasm, which have even a more unfavorable prognosis than the varieties we have hitherto considered, and which, according to their special manifestations, are classed as *febris intermittens eclamptica, epileptica, tetanica*, and *hydrophobica.*

The *eclamptic* form is found mostly among children and puerperal women, and often begins, as does the epileptic form, with headache, lassitude, restless nights, and not seldom with delirium, to which the convulsions are then suddenly added, often beginning with a piercing yell, followed by complete loss of consciousness and sensibility, with dilated and immovable pupils. *Tetanic spasms*, undoubtedly the most pernicious variety of the disease, and known to the ancients under the name of katochus, are comparatively rare. They sometimes begin with weakness in the legs and convulsions of the extremities, or with trismus, and are not infrequently accompanied with delirium. The patient lies unconscious and insensible, with a red countenance, and frothing at the mouth, an irregular pulse, the hands clenched, the jaws set, the neck stiff, and the body bent either forward or backward. Sometimes opisthotonos and emprosthotonos alternate, pleurosthotonos being of less frequent occurrence.

Still another form of the disorder has been found sometimes, generally amongst women, and causing serious psychical disturbances, which has been designated as *intermittens hydrophobica.*

It manifests itself as violent maniacal delirium, with a frequent pulse, red, glowing face, and clonic spasms of the muscles of deglutition on drinking, or even at the sight of water; these spasms then pass to the muscles of the face, the eyes, and the neck, and finally to those of the entire body, a disposition to bite being at the same time developed. These attacks usually last some hours, passing gradually into a state of sopor, and may be repeated several times, according to some regular type.[1]

Aside from these severe nervous disturbances, intermittent fever may assume a pernicious character by virtue of other alarming symptoms. Under this head comes the *choleraic form* of the malady, which is of quite frequent occurrence, and in which burning thirst, severe vomiting of yellowish matter, and greenish, diarrhœal passages appear during the continuance of the fever,—the passages from the bowels becoming more and more watery, and sometimes resembling bloody water. Subsequently, just as in real cholera, general oppression and the most tormenting præcordial distress are felt; cramps in the calves of the legs, coldness and a livid appearance of the skin follow, with a small, hardly perceptible pulse, sunken eyes, and a true Hippocratic countenance. Convulsions are not infrequent. Death supervenes with all the evidences of asphyxia. Another variety that bears a striking resemblance to the foregoing is that known as *algid intermittent*, characterized by a coldness of the body like that of marble, while the temperature in the mouth is from 86° to 88° Fahr., and that in the axilla 84°. The attack begins with the usual chill, which is not of abnormal severity or duration. It is not until during or after the hot stage that the patient grows cold, while at the same time he complains of burning heat within, and of thirst; the skin turns pale and livid, is stiff and covered with a cold sweat, the pupils are dilated, the eyes expressionless, the lips blanched and dry, the tongue smooth, pale, and soft, and the region of the stomach sensitive. Muco-bilious vomiting is liable to occur, with occasional greenish-yellow stools, though both of these may be absent, and are of subordinate importance. The urine is scanty, dark-colored, turbid, of high specific gravity, and free from albumen; the pulse is irregular, often much retarded, as slow as forty in a minute, small and thready; the respiration is shallow, as slow as ten in a minute; the voice feeble, hoarse, and broken. Consciousness is preserved intact, though the patient is apathetic, and there is nothing in his condition to wrest from him any outcry or complaint.

---

[1] *Waton*, Gaz. méd. de Lyon, 1857, No. 6. Canstatt's Jahresber., 1857, IV, p. 178, describes two such cases.

This form of pernicious malarial fever is usually confined to warm countries, and is, as a rule, preceded by some paroxysms of simple intermittent. Even in these simple paroxysms, however, according to Rigler,[1] there is an unusual prostration, reaction is imperfect, and the coldness of the face and extremities during the paroxysm is striking. It is said that in exceptional cases these attacks may terminate in spontaneous recovery; usually, however, death takes place, either in the first attack, or, if they are repeated, in the second to the fourth. Recovery follows through a reaction, like that from cholera; it is not unusual, either, to find a typhoid condition supervene, like that after cholera. Convalescence is said to be slow, accompanied with headaches, dizziness, wakefulness, irritability of temper, and partial loss of memory. In case of death, the patient usually maintains his consciousness to the very last; while the pulse grows smaller, slower, and more irregular, until it entirely disappears, and the chest expands more and more feebly, until, at last, the heart and respiration come to a complete stand-still.

---

[1] Wiener Wochenschrift, 1858, No. 18.

In connection with the preceding, we may consider another form but little known, and designated as *febris perniciosa diaphoretica*, in which a very colliquative and continuous sweat appears at the end of the stages of chill and heat, lasting during the succeeding intermission, and producing great coldness of the surface. The skin assumes a waxen hue, the lips and the buccal mucous membrane grow pale, insatiable thirst and great prostration supervene, the heart's action becomes feebler, respiration more labored, and the patient dies.

What is known as *febris perniciosa cardialgica* is accompanied with severe burning, darting, contracting pains in the region of the stomach, during the stage of chill, with nausea, retching, and vomiting; great anxiety and restlessness; a red, dry tongue; the skin being at first cold, afterwards hot and dry. This form, it is said, after passing through the stage of sweating, usually ends in recovery; still, some claim that the latter paroxysms, if there are such, may terminate fatally. In *febris perniciosa dysenterica*, a localization of the malarial disease in the intestinal canal, colic and tenesmus, as a rule, appear suddenly during the febrile attack, with very copious dejections, first of a serous character, afterwards consisting of blood and mucus, or of pure blood; these passages cease after the attack is passed, to reappear again during a subsequent paroxysm. Such attacks, which may also sometimes be associated with cerebral symptoms (Bierbaum), present themselves in the quotidian or tertian type, and during the intermission the dejections are bloodless and normal.

It sometimes happens that serious *hemorrhage* from the *stomach* or *bowels* takes place during one or another stage of a paroxysm, endangering the life of the patient, or leading to deep fainting fits. Cases of typical gastritis have also been observed, in which, during the hot stage, violent vomiting resulted in the rejection of a brownish or bloody fluid, while at the same time there were copious watery passages from the bowels, and the abdomen was distended and very painful. Waton[1] saw one case in which the symptoms of peritonitis were developed at seven o'clock in the evening;—intense pain in the abdomen, vomiting, a haggard countenance, and a small, compressible pulse.

Aside from the cases in which a pre-existing bronchitis, pneumonia, or pleurisy is aggravated in its symptoms or modified in its course by an intercurrent malarial fever, it also appears possible for such a fever to localize itself in the bronchial tubes, the lungs, or the pleura, and there to call forth manifestations in no degree inferior to those of the genuine diseases of these organs. We may thus meet with a sudden outbreak of hyperæmia of the bronchi, in which the patient, with livid countenance and distended jugular veins, suffers from the most intense anxiety and constriction of the chest. Or, in persons predisposed thereto, especially those who suffer from frequent congestions of the lungs, or give evidences of scurvy, the most violent pulmonary hemorrhages may ensue, which, if often repeated, reduce the vital powers to the utmost, and may result in death. More interesting than these, however, are the cases of *intermittens perniciosa pneumonica* and *pleuritica*. During the chill and the hot stage, the patients are seized with dyspnœa, they cough much, without expectoration, and complain of the most violent pain in the breast. The pulse soon becomes full, rust-colored sputa appear, dulness on percussion and increased vocal fremitus are manifest, while auscultation reveals, at first crepitant rhonchi, then bronchial breathing and bronchophony. During the sweating stage a decided remission of the fever takes place, as well as an abatement of the symptoms, subjective and objective, which may almost entirely disappear during the intermission that follows. In case of a renewed paroxysm, however, of the quotidian or tertian type, these symptoms will recur. The infiltration will now increase somewhat in extent, and remain stationary during the intermissions. Griesinger compares the filling up of the lung that takes place under the paroxysms of malarial fever to the enlargements of the spleen, which at first recede, during the intervals of the fever, but afterwards remain constant. The lower lobe of the left lung appears to be the one generally involved.[1] If timely recourse to treatment by quinine is neglected, these cases are apt to end fatally at about the fourth or sixth paroxysm.

---

[1] Gaz. méd. de Lyon, 1857, No. 6.—Canstatt's Jahresb., 1857, IV, p. 177.

Some years ago I had the opportunity of observing a case of *intermittens perniciosa pleuritica*, which is even more rare than the pneumonic form. The subject was a very powerful man, thirty-eight years of age, who was seized, about two o'clock at night, with slight chilliness, very severe pain in the left side of the chest, and a dry cough. Towards eight in the morning, while the symptoms were all present in the highest degree, I discovered a distinct, rough, friction sound, the temperature being moderately elevated and the skin hot. Wet cups and an opiate were ordered, and I was not a little surprised the next morning to find the man entirely free from pain and from all tendency to cough, and nothing wrong to be discovered on auscultation. At twelve o'clock that night, however, the attack was repeated. At nine in the morning I found the same symptoms as two days before, although in greater intensity, and the friction sound was not only more plain but more widely extended. As I found the patient, at 4 P.M., bathed in perspiration, almost free from fever and from pain, and as I found him well again the next morning, I ordered a few large doses of quinine, whereby the disease was cured without another attack.

It remains for us to notice the *icteric* form of malarial fever. This is usually developed from a simple intermittent, and prevails endemically in certain localities, as on the island of Madagascar[2] and at Nosse Bé.[3] It often seems to give certain warnings of its approach, twelve to twenty-four hours in advance, such as headache, lassitude, nausea, chilliness, and sometimes a slight icteric coloring of the conjunctiva or of the alæ of the nose. The actual attack always begins with a violent, long-continued chill, during which the icteric color either first begins, or, if already present, grows more intense, spreads over the entire body, and soon assumes a saffron or olive hue. Intense nausea is now felt, with copious vomiting of bile, and often bilious diarrhœa. Thirst is urgent; the tongue is coated white and yellow, and the patient suffers with excruciating headache, pain in the spleen and over the liver, and a feeling of numbness in the limbs. The pulse is small, frequent, and hard; the urine is of a deep red color, resembling porter or Malaga wine, though free from blood, and owing its color to the abundant presence of biliary coloring matter; it is quite scanty, and the effort to void it causes distressing tenesmus. During the hot stage the pulse grows fuller and more frequent, the respiration anxious and noisy, the skin hot, and the thirst more tormenting. Bilious diarrhœa and vomiting continue, and the urine grows still more scanty. During this stage, which lasts from three to five hours, death often occurs; otherwise the disease passes on to the third stage, or that of sweating. The skin is now bathed in a profuse perspiration, which, like the urine, stains the linen yellow, and the symptoms gradually improve. During the intermission the head is generally clear and the intelligence perfect; the icterus persists, although the urine regains its natural color. With proper treatment recovery may follow, though an unfavorable termination is, even then, frequent. In that case the intermissions are not complete, the disease showing a tendency to assume the remittent or continued type. All the symptoms are aggravated in the subsequent attacks, vomiting continues without interruption, the bowels and bladder are emptied involuntarily, the patient gradually sinks into coma, and death usually follows in the second or third paroxysm—seldom later.

---

[1] A case recently occurred in my clinic, in which the lower portion of the right upper lobe was the part involved.

[2] *Daullé*, Gaz. des Hôp., 1858, No. 3.

[3] *Faymoreau*, Canstatt's Jahresber., 1860, IV, p. 78.

Malarial fever may have a pernicious character given to it by the occurrence of *gangrene* in some portion of the body. Scholz reports one case of death in a man, from gangrene of the leg, and one in a woman, from gangrene of the genitals. Poumier saw gangrene of one leg developed in a woman who had suffered four or five days before from intermittent fever; and Lafaye reports the case of a girl, five years old, one of whose hands grew black and gangrenous after a few paroxysms of malarial fever; two days later, on the occurrence of a new attack, the other hand became similarly involved.

Some years ago a man about twenty-five years of age, a worker in the "polders," was brought to my clinic at evening in a comatose condition, the result of a malarial attack. He had been the subject, a few days before, of two ordinary attacks of intermittent fever. Large doses of quinine, stimulating enemata, and derivation to the skin brought about a rapid recovery; but on changing his linen the next morning a bluish-red spot, about as large as the palm of one's hand, was discovered in the right gluteal region, which became gangrenous in a few days, and penetrated to an alarming depth. He had no more attacks of intermittent fever, but was obliged to remain in hospital several weeks for the cure of his bed-sore.

Remittent and Continued Malarial Fevers.

This class consists of fevers which present no intermissions or apyrexias during their course, but only more or less marked remissions and exacerbations; but which must be reckoned as amongst the diseases due to malarial infection, because of their frequent transition into ordinary intermittent fever, their anatomical lesions, their occurrence in malarious regions, and the fact that the same remedy is required for the cure of both conditions.

Remittent and continued fevers prevail chiefly in the most intensely malarious regions, especially in the tropics, as well as in more northerly latitudes during the spread of a powerful epidemic. They also occur sporadically, and in a mild form, in the latter regions, during the hot months, especially after heavy rains and inundations.

According to Griesinger, the topographical distribution of these forms of the disease seems to differ from that of simple intermittent, in districts where malaria is endemic; inasmuch as the remittent variety prevails in the humid coast regions, and ordinary intermittent on higher lands, a circumstance undoubtedly due to the varied intensity of the morbific agent. When, as is frequently the case in epidemics, we see, now the remittent, and again the intermittent form prevail, without any special, demonstrable cause, we are justified in attributing this also to the varying intensity of the poison; just as we often see epidemics begin with the intermittent form, change to the remittent while at their height, and return to the intermittent again during their decline. Where both forms coexist within a given area, individual predispositions and peculiarities of constitution may occasion the development of the one or the other in a given person.

There are various ways in which intermittent fever may assume the remittent or continued type. The intermission may be shrouded, or may be made completely to disappear, merely through the persistent anticipating of the paroxysms; or through a relatively long duration of the same as compared with the usually brief apyrexia, due to the simultaneous existence of some other febrile affection, such as gastric disturbances, bronchitis, pneumonia, etc.

The remittent forms of disease, however, so far as they are to be regarded as a distinctive class, show some peculiarities, other than the mere absence of an intermission, which are entitled to special mention, and this is particularly true of those varieties which are peculiar to the tropics. But not all those diseases which are regarded as remittent or continued malarial fevers, in the tropics or in more northern malarial districts, could maintain their claim to this title if subjected to the test of scientific criticism. Even the physicians of our own lands are too liable to designate as malarial diseases attacks of pneumonia accompanied with jaundice, or simple gastric disturbances, with or without jaundice, or mild grades of typhoid fever running an irregular course.

The pernicious character of remittent fever either shows itself from the very beginning, or appears on the third, fourth, or sixth day, or later, even as late as the second week. The exacerbations usually recur in the quotidian, more rarely in the tertian type; they are sometimes patent, sometimes hidden, and only to be discovered by careful observation and thermometric observations.

The division of remittent and continued fevers into those of *light*, *heavier*, and *highest grades*, as it is made by Griesinger, finds its justification in the fact that it isolates certain pictures of disease, and facilitates our study of their features. But we must never forget that it is not intended thereby to designate distinct forms of disease, for an interchange in the several grades of intensity is frequent, and, in some epidemics, the rule.

The *first grade*, that of the *lighter forms*, embraces the bilious or gastric remittents, which often appear in great numbers during a hot summer, in malarious districts, associated with gastro-intestinal catarrh and icterus. It not infrequently happens, as I have observed in Holland, that the actual attack is preceded for several days by gastric disturbances, loss of appetite, nausea, irregularity of the bowels, with occasional irregular rigors and dizziness. Usually, however, the attacks begin suddenly, and without warning, with a moderate chill, followed by loss of appetite, nausea, the raising of sour material from the stomach, a general feeling of sickness, with severe headache, dizziness, ringing in the ears, sleeplessness, and restless nights. Jaundice and a colorless diarrhœa follow afterwards. Sometimes an herpetic eruption is developed about the lips; bleeding at the nose is not uncommon, neither is a light grade of bronchitis. With all this, sensitiveness is developed over the region of the spleen, with an enlargement of that organ not difficult of demonstration. The disease usually reaches its end in three, five, or ten days. More rarely it does not terminate for some weeks, and then it runs the following course. The exacerbations which occur daily, twice a day, or every other day, and which are not infrequently ushered in by a new chill, are followed by more and more distinct remissions, with sweating and an abatement of the general ill-feeling, these remissions not seldom passing, by degrees, into actual intermissions. I have occasionally seen patients in whom, after the temperature had fallen to its normal standard, the gastric disturbances would persist for several weeks, accompanied by no small degree of jaundice.

The *second grade*, that of the *severer forms*, is especially characterized by a more continuous course, with only here and there marked remissions, especially at the beginning of the attack. Although beginning as a sthenic fever, they not infrequently early assume a typhoid or adynamic character, and may, aside from the greater intensity of the general disease, be associated with particular and pernicious local affections, as with dangerous nervous symptoms, with delirium, with eclamptic, epileptic, tetanic, paralytic, or comatose seizures; furthermore, with algid and dysenteric conditions, as well as with parotitis, pneumonia, and inflammation of the spleen, all of which are likely to end in death.

The disease usually begins with severe headache, delirium, and a heavy chill. The primary disease, when not complicated with any of the conditions mentioned above, lasts, according to Griesinger, from seven to fourteen days. Recovery follows, with marked remissions and sweats, or by transition to a simple intermittent, although relapses are said to be frequent.

The *third grade*, that of the *severest forms*, depends, according to Griesinger, either upon specially unfavorable constitutional or external conditions (debilitating treatment, septic influences, etc.), or on the too great intensity of the pernicious features. They are particularly distinguished by the most marked and extreme adynamic character, by a tendency to rapid collapse, and by the early condition of apathy into which their victims fall.

These attacks sometimes begin in a mild form, soon, however, showing indefinite and irregular exacerbations and remissions, the former of which become constantly shorter and the latter longer and more exhausting, with the temperature of the body below the normal standard, thus indicating the adynamic character of the disease. Patients complain of pain in their limbs and joints, with difficulty of moving the same; but the feeling of pain is soon blunted, sight is sometimes dimmed, memory is weakened, drowsiness and stupor are prominent, or, very rarely, delirium and wakefulness. The patient falls into a state of mental apathy, and death usually follows while he is in an algid and comatose condition. At other times the disease shows the gravest symptoms from the very commencement, beginning with a chill and heat, followed in a few hours by a state of complete exhaustion, with disturbed intellect, which state is sometimes accompanied by great restlessness and delirium. Respiration is apt to be sighing; the pulse small and rapid, 100 to 140; the tongue coated white, or clean, pale and rough; the skin hot and dry; the abdomen distended and painful; thirst extreme, and the spleen enlarged and sensitive. There is frequently vomiting, and the strength gradually declines, so that death, through exhaustion, may occur at the end of five or six days. In a malignant epidemic which was observed by Gibbs[1] on the west coast of Nicaragua, in May, 1868, the attacks were never introduced by a chill. Death occurred within a few days, at the height of the disease, without any reaction having followed, the symptoms being severe headache, delirium, yellowness of the conjunctiva, continuous severe vomiting, a cool skin, a bad and sometimes slow pulse, and deep collapse. Occasionally, during the course of the disease, other pernicious local symptoms are developed, such as enormous swelling of the liver or spleen, with the occasional formation of abscess, purulent collections in the serous cavities, pneumonic infiltration, icterus of the skin, partial œdema, bed-sores, gangrene of external parts, petechiæ, epistaxis, hæmoptysis and hæmatemesis, or dysenteric and choleraic dejections.

Most of the cases of this severest form of remittent fever terminate fatally; the few patients that recover remaining weak and sickly for a long time.

In the above sketch I have merely delineated some of the leading types of the disease in question; these could, without difficulty, be largely increased, for almost every epidemic shows some variations in form peculiar to itself. I shall only permit myself, however, to notice one special form, which, although long known, has not met with general recognition until of late years, when it has been most carefully observed and described.[2] It is that affection called by the French, from its prominent symptoms of a bilious condition and bloody urine, *fièvre bilieuse hématurique.*

This disease is found, as a rule, only in the most intensely malarious portions of the tropics, and then only at the height of an epidemic, during and immediately after the rainy season; still it may occur in more northerly latitudes, and has even been met with in Holland at the time of a malignant epidemic. Of late years, so far as reports have been received, this affection has been endemic in Senegambia, at Mayotte in Madagascar, in Cochin-China, Cayenne, on the Lesser Antilles of Guadeloupe and Martinique, in various portions of Alabama, Louisiana, Arkansas, Florida, Texas, and along the banks of the Mississippi. It is usually developed in persons who have been exposed to malarious influences for a long time, and have often suffered with intermittent fever, or in whom a true malarial cachexia has been established.

---

[1] Virchow's and Hirsch's Jahresber., 1868, II, p. 199.

[2] *Barthélemy-Benoit*, Arch. de méd. nav., 1865, p. 5. *Veillard*, De la fièvre bilieuse hématurique. Paris, 1867. *Hirsch* and *Virchow*, Jahresber., 1868, II, 200.

It has always been a matter of doubt whether this affection should be recognized as an independent form of disease or be considered merely as a modification of some other familiar forms. Some writers consider it identical with the icteric pernicious fever, others look upon it as a modification of yellow fever, while still others believe that it occupies a middle ground between common malarial and yellow fever. The disease ordinarily begins as a simple intermittent, the symptoms of which, however, undergo an unusual aggravation after the occurrence of a few paroxysms. They then manifest themselves in the form of severe, long-continued chills, severe pain in the head, back, and loins, burning pain in the pharynx, the œsophagus, and the region of the stomach, urgent thirst, nausea, and persistent vomiting of biliary matter. At first there is usually obstinate constipation, and afterwards, bilious diarrhœa. The urine is usually voided in great abundance, is dark brown, and rich in blood. Veillard was never able to demonstrate the presence of biliary coloring matter in the urine. The skin is slightly jaundiced and covered with a cold sweat, the features are sunken, and the pulse grows gradually smaller, running at the rate of from 80 to 130 beats. The liver and spleen are greatly enlarged. The fever assumes the remittent or continued form. Under early and appropriate treatment, and with a patient of good constitution, recovery is possible; usually, however, coma supervenes as early as the second day, or, it may be, not until the fourth to the sixth day. The pulse grows smaller, becoming hardly perceptible, dyspnœa increases, the urine becomes scanty or is entirely suppressed; then ecchymoses appear, hemorrhages from the nose, the lungs, and the stomach; and death ensues, usually during the second week.

Chronic Malarial Infection, or Malarial Cachexia.

Although the condition known as *malarial cachexia* is usually classed among the sequels of ordinary malarial disease, yet there are reasons that justify us in taking up its consideration at this time. For not only is this condition developed after frequent repetitions of simple intermittent, or in case of the long duration or neglect of, or incomplete recovery from the same, or where certain organic diseases or anomalies of constitution exist, or after severe attacks of remittent fever; but a primary chronic affection is very often observed, in malarious regions, without any antecedent attacks of malarial fever, and this primary infection, after having existed for a long time, often comes gradually to present symptoms similar to those of the secondary cachexia. These primary affections are only to be met with in the most intensely malarious regions, and it is not going too far to assert that most of the inhabitants of such regions actually do suffer from this infection without its having been preceded by any outbreaks of intermittent fever.

In the interior provinces of Holland this state of things is familiar to every one, and is designated by the vulgar name of "binnen koorts," inward fever. It betrays itself by the most varied manifestations, changing frequently in the same individual, often showing a tendency to assume a definite type, and involving certain nerve-tracts, so that for a long time I was in doubt whether some of these cases were not, properly speaking, masked intermittents. The paroxysms, however, are only feigned, and their apparent periodicity depends upon the fact that, owing to certain injurious influences affecting the individual, some single symptoms are rendered more prominent at given hours of the day. In general, however, the course of the affection is continued.

During ordinary times, when no intense malarial diseases prevail, the persons attacked are usually those who have been continuously exposed to malarial poison, or in whom certain incidental causes have been at work, such as imprudent methods of living, damp, low dwellings, or bedchambers, etc., or those who for some unknown reason present a special predisposition thereto. Great heat and a damp atmosphere exert their influence here also, and increase the amount of the disease.

Patients thus affected complain of dizziness, a symptom which, according to Schramm, is so general in his neighborhood that there are but few people not familiar with it; they also have ringing in the ears, muscæ volitantes, and disturbances of visual accommodation; they lose their appetite, have a metallic taste in the mouth, nausea, and a disposition to vomit, with eructation of gases; there is dryness of the lips, mouth, and throat, the tongue, in the middle and behind, being coated with a yellowish-white fur. Sometimes there is an increased secretion of saliva, roughness in the throat, and a disposition to clear it, which usually produces nausea and retching. A feeling of pain and oppression in the epigastrium is frequent, especially when the stomach is empty; also rumbling in the bowels and a frequent desire to go to stool, with or without tenesmus. The condition of the bowels is usually normal; constipation is rare, and oftener there are several diarrhœal passages during the day, generally during the morning hours. Patients perspire quite copiously after every little movement of the body, and during the night. Sleep is often disturbed, and even interrupted for hours, or it may be very profound but unrefreshing, the individual feeling in the morning as if he had been up or had travelled all night, with his head confused and a general feeling of discomfort. Many complain of tickling and burning in the feet and hands, of formication and a sensation of deafness, of pain in the shins, or of dragging pain in the course of the sciatic nerve, in the loins, in the small of the back, and over the coccyx, which latter is also painful on pressure, and after prolonged standing or walking becomes the seat of the most unendurable burning. Others mention pains in the joints, often of the knee-joint, or in the muscles of the extremities, or they experience stiffness in the limbs and the back, become easily fatigued, especially in climbing stairs, are short of breath, and have palpitation of the heart. The complexion quickly assumes a yellowish, pale hue, the conjunctiva is of normal color, as is also the urine, which is often excessively abundant, and in that case as clear as water, of low specific gravity, and without any abnormal constituents. With many patients, temporary attacks of melancholy arise from time to time; with others the hypochondriac condition is a constant one. All such imagine themselves very ill, they suspect disease of the heart, of the spine, or of the kidneys, and in one of my patients even a light form of agoraphobia was developed, which yielded to quinine.

I have never found the temperature to be elevated in this condition; the pulse is normal, or a little rapid, or, in many cases, a little below the average frequency. The spleen is generally enlarged, and patients often complain of sensitiveness in the left hypochondrium on pressure or when they cough.

This light form of malarial intoxication often disappears spontaneously as soon as the predisposing and exciting conditions are removed; it is quite sure to disappear under the use of quinine, but, as in all malarial affections, relapses are frequent. Other patients, in whom the infection continues, and these means are neglected, may suffer with it for weeks and months, and it may gradually pass into the more severe forms. It also not infrequently happens that attacks of true intermittent vary the course of the disease. Ritter,[1] who practises in the Hannoverian moorland colonies, and to whom we are indebted for some interesting reports on malaria, likewise describes a primary chronic malarial infection, which begins with headache, loss of appetite, a bitter or salty taste in the mouth, lassitude, pain in the limbs and back, a coated tongue, and constipation, and which eventually brings the patient to his physician with some complaint, whether of the alimentary canal (gastric or intestinal catarrh or salivation), of the respiratory organs (chronic bronchial catarrh), or of the nervous system; for the constant disturbance in the head finally brings the individual into a hypochondriac condition. At the same time, Ritter's description of the subsequent course of these attacks resembles, in many respects, that which I shall give as belonging to the *severe forms* of primary infection.

---

[1] Virchow's Archiv, Bd. XXX, p. 273.

These severe forms also begin with a diminution of appetite, with sluggish digestion and constipation, which may soon be followed by uncontrollable diarrhœa and frequent vomiting—in graver cases by all the symptoms of dysentery. Patients are easily fatigued and exhausted, and get short of breath, the complexion is pale and grayish yellow, and the conjunctiva slightly jaundiced. The urine is scanty, dark-colored, free from albumen, and sometimes contains a considerable amount of biliary coloring matter, in which case the skin and conjunctiva are icteric and the fæces colorless from the absence of bile. The spleen is but very rarely of normal size, it is generally enlarged, and indeed to such a degree, as to half fill the cavity of the abdomen ;[1] the liver also may show an immense enlargement ; both organs are hard and sensitive. The abdomen is greatly distended by the enlargement of these organs and by meteorism. Ascites is developed, as well as œdema of the face and extremities, with a tendency to sores ; furuncles appear in the skin, and portions of the body may become gangrenous. Patients complain of palpitation of the heart, the pulse is feeble, though not frequent, a venous murmur may be heard in the neck, and blood-murmurs in the heart. Hemorrhages from the nose are frequent, those from the female genitals, or from the stomach and bowels, rare ; petechiæ appear upon the skin, and a scorbutic condition is sometimes developed about the gums. It is claimed that in very severe cases, such as I have never seen, profounder disturbances of the nervous system arise, such as trembling, choreic movements, paralyses, delirium, and disturbances of vision and of intelligence. All these symptoms show no periodicity ; nor can any rise of temperature be demonstrated at any time, unless, indeed, what is not infrequent, regular attacks of intermittent fever supervene, which then assume the usual quotidian, tertian, or quartan type. Sometimes this type is not assumed, and the disease maintains the character of a remittent or continued fever.

---

[1] *Faymoreau* (Canstatt's Jahresbericht, 1860, IV, p. 74) observed inflammatory processes, and in three cases fatal suppuration of the spleen.

The train of symptoms just described is the same that is encountered in malarial cachexia when it follows previous intermittent fever.

Mild grades of chronic infection and cachexia may recover under appropriate treatment; in the higher grades, however, death generally results through exhaustion, with dropsical manifestations, renal diseases, amyloid and leucæmic conditions; or, it may be, through the addition of tuberculosis, dysentery, pneumonia, or pleurisy. Patients sometimes die of apoplexy, which Griesinger thinks may be due to a pigment embolism of the cerebral vessels.

Pathological Anatomy.

The fact that we know so little of the anatomical changes which take place in *simple intermittent fever* is evidently in part explained by the circumstance that hardly any such patients ever become the subject of a post-mortem examination, unless their death is brought about by some intercurrent disease. When such complications exist, it is not always easy to determine what proportion of any lesions that may be found depends on the intermittent, and what on the other disease.

With regard to the primary *blood changes* which take place in primary malarial infection, we are, thus far, without any reliable chemical or microscopical data; for the deviations from their normal composition which we recognize in the serum and the blood-corpuscles occur only as the result of the disease, that is, they depend on the consumption of tissue caused by the fever and the high temperature, as well as on the changes produced in the spleen and in other important organs. For, even in recent cases of simple intermittent, the spleen is, with few exceptions, enlarged by simple hyperæmia; in fact, tumors of the spleen have been observed in the newly born children of mothers who suffered from malarial fever during pregnancy; but structural changes in this organ occur only when the paroxysms of fever are very often repeated or of long duration. The same thing is true of masked fevers; for, in spite of the manifold localizations of the same, no one has, as yet, succeeded in demonstrating any palpable changes in the nerves or the organs affected, except in those cases where it was a question of visible hyperæmia, œdema, or inflammatory disturbances of nutrition.

The results of minute anatomical investigation have been more satisfactory in the severer forms of malarial disease, in the *pernicious*, *remittent*, and *continued* fevers, as well as in the higher grades of *primary* and *secondary malarial cachexia.*

A very frequent condition, in the severe forms of malarial fever, is that characterized by the presence of a dark brown or black pigment matter, in the form of fine granules, or of cells and flakes containing pigment granules, which are found in the blood, in the spleen, the liver, the brain, the spinal cord, the kidneys, especially the cortical portion, the heart, the lungs, and the lymphatic glands; also in the integument, in the serous membranes, etc., showing themselves here by characteristic, usually slate-colored discolorations in the course of the blood-vessels. The spleen has long been supposed to be the point of origin of this pigment matter, and it has been thought to be distributed thence after the manner of emboli. There seems, however, to be some evidence to prove that the liver is not without a share in the production of this pigment; while others believe that it is also produced in the blood, as the frequent considerable size of the pigment flakes in the vessels of the brain excludes the possibility of a passage through the capillary system of the liver and lungs. The origin of pigment matter in the spleen has been thought to be due to the considerable fluxionary hyperæmia occurring during the fever paroxysms, by the escape of blood-corpuscles through the sound or ruptured walls of the vessels; or, according to Duchek, by the arrest of the circulation in very many of the venous sinuses. In view of his observations to the effect that the pigment matter is not found in the tissues of organs, but in the walls of blood-vessels, Heschl[1] believes that, as the hæmatine is retained in the corpuscles by virtue of the presence of a certain amount of albumen and common salt in the plasma, so changes in the plasma are the cause of the escape of the hæmatine, which changes are produced by the malarial poison that has penetrated the body. The greater collection of pigment matter in the capillaries of the spleen, the liver, the brain, and the spinal cord, leads Heschl to suppose that the poison operates on these organs in particular; that it there causes a disintegration of blood-corpuscles, and that the hæmatine, which is now set free, and which becomes a carrier of the poison, penetrates the walls of the capillaries.

---

[1] Oestr. Zeitschr. f. prakt. Heilkunde, 1862, No. 40 et seq.

In the bodies of those who have died of pernicious malarial fever, the *nervous system* often gives evidence of no change at all, or of but very slight changes, notwithstanding the grave symptoms that occurred during life. In the comatose form, a brownish or slate-colored discoloration of the cortical portion of the brain, and sometimes also of the ganglia, has occasionally been discovered, which was due to accumulations of pigment matter, as well as numerous punctiform extravasations, which have been thought to depend on a blocking up of the capillaries by means of pigment, but which Heschl says may exist independent of the formation of pigment matter, and represent capillary aneurisms. Instead of this, however, it is more common to find only more or less hyperæmia and œdema of the brain substance and its membranes, together with an accumulation of serum in the ventricles. It is rare to find extravasations of any size in the brain or its membranes, or in the midst of centres of softening.[1] The same state of things is found after the delirious form of the disease—indeed hyperæmia and œdema are the principal conditions present after all the disturbances of the central nervous system connected herewith. In the delirious form, indications of the exudation of coagulable lymph into the membranes of the brain have been observed. In the form characterized by tetanic cramps, and formerly called katochus, Girard saw softening of the brain and of the upper part of the spinal cord, in addition to hyperæmia. In the algid form the substance of the brain and cord is pale, bloodless, dry, and tough. In cases of icteric intermittent the membranes have been found colored yellow, with a yellowish fluid effused into the ventricles and under the arachnoid. The *lungs* are often highly hyperæmic and œdematous, not seldom showing hemorrhagic infarctions, and in the pneumonic form red and gray hepatization. The *heart* is usually dilated, its walls flaccid, and sometimes pale. In the algid form the endocardium and pericardium are ecchymotic, the muscular substance dark, infiltrated, tender, and easily torn. In one case Rigler found several extravasations as large as a pea. The right side of the heart and the venæ cavæ are filled with abundant quantities of dark, half-clotted blood. In most cases of pernicious intermittent, the *spleen* is enlarged, reaching three or four times its normal size; it is hyperæmic and softened, sometimes consisting of nothing but a sac filled with a black, bloody fluid. Infarctions are not very rare, which may result in gangrene or the formation of abscess (cases reported by Scholz, Duchek, and myself), and rupture of the spleen, with fatal hemorrhage into the cavity of the abdomen, may occur. It is not uncommon to find discolorations and thickening of the capsule of the spleen, and adhesions to neighboring organs. Under some circumstances, when death occurred during the first or second attack, the spleen has been found unchanged, this condition being particularly observed by Girard in the malignant fevers prevalent at Rome. The *liver* is often enlarged, in acute cases hyperæmic, the parenchyma swollen and soft; sometimes, especially in the pernicious fevers of the tropics, it is studded with numerous apoplectic spots, or with disseminated and confluent inflammatory and purulent collections. In the icteric form the organ is very large, the gall-bladder usually filled with thick, dark-brown bile. The *intestinal* mucous membrane is either simply hyperæmic, and shows the changes incident to a catarrhal condition, or it is ecchymotic, as in the choleraic cases. The solitary follicles and Peyer's patches in the small intestine are often greatly swollen, and colored with pigment matter; in the icteric form they are filled with a bile-colored fluid; the colon is swollen, and highly hyperæmic, its follicles are infiltrated and partly suppurating, or the mucous membrane shows extensive dysenteric processes. The mucous membrane of the *stomach*, too, is often hyperæmic;

---

[1] *Titeca*, Arch. méd. belges, 1869, Jan., p. 5, and Juin, p. 403, found, in two cases, red areas of softening in the brain of from two to three centimetres in size, without any demonstrable cause of capillary obstruction.

in those cases that have had hæmatemesis it is highly ecchymotic. The *kidneys* are sometimes swollen, the mucous membrane of the hilus being hyperæmic. The integument is generally of a pale yellow, and in icteric cases of a deep yellow color. In case of the throwing off of pigment matter it becomes of an ashy gray.

The anatomical changes in the severe forms of *remittent* and *continued* fever, as far as observations are collected, show a great conformity to the picture just drawn of the conditions in pernicious intermittent. The fact of the deposit of pigment matter in various organs, in this form too, as has been verified by many observers, is an argument in favor of the view that all these processes are only modifications of the same fundamental disease. The other changes, too, in the spleen and liver, the swellings and enlargements, the processes of softening and suppuration, the catarrhal, hyperæmic, and follicular changes in the intestinal canal, the relaxation and discoloration of the muscular substance of the heart, etc., all these only go to confirm the above hypothesis. With regard to *adynamic* remittent fever, it may here be stated that, according to Brown, Stewardson, and others, the hyperæmia, so commonly found in other forms, is lacking; on the contrary, the brain, liver, spleen, heart, and all the voluntary muscles are distinguished by great relaxation and softening, the extreme softness and ease with which the tissue of the heart would tear being noticeable, as well as the watery quality of the blood, and its evident deficiency in fibrine. In the autopsies made of those who died of the *fièvre bilieuse hématurique*, there was generally very considerable hyperæmia, besides swelling and softening of the spleen and liver, the latter being strongly icteric and sometimes fatty; the dark-colored skin was sometimes the seat of petechiæ, ecchymoses, and pustules filled with blood (Veillard); the brain and its membranes were rich in blood, and icteric; the gall-bladder contained thick, black bile; the mucous membrane of the stomach and bowels was very vascular and soft; the heart soft and relaxed; the kidneys highly hyperæmic; the bladder usually empty, injected, and ecchymotic, the urine always containing blood and albumen; still the question of the presence of biliary coloring matter in the urine is as yet unsettled, some observers accepting it as a fact, while others, like Veillard, have never been able to demonstrate its presence.

In considering the anatomical changes which take place in *malarial cachexia*, and after long-continued and oft-repeated attacks of intermittent fever, we find, in addition to an anæmic color of the skin, to œdema of the subcutaneous cellular tissue and the accumulation of fluids in the serous cavities, to atrophy of the muscular and adipose tissues, etc., certain alterations in the liver, the spleen, the kidneys, and the heart, which claim our further attention. The "*ague-cakes*," with which every one has long been familiar, are the results of frequent relapses of intermittent fever, lasting for a long time, and of primary chronic malarial infection of a high grade. They consist of firm, tough, slaty tumors, presenting a smooth surface on section, rich in pigment matter, of enormous size, often fifteen or sixteen times the natural size of the organ, of the shape of a cake, usually provided with a thickened capsule, and attached by adhesions to the surrounding parts or organs. They represent either simple hyperplastic conditions or amyloid degeneration. Leucæmic tumors of the spleen seem to be of more rare occurrence, inasmuch as not a single case of the kind fell under my observation in Holland during six years; and Tomasi, during four years, within which time he saw very many tumors of the spleen, could never demonstrate any one of them to be leucæmic. Occasionally the spleen may diminish in size again after having been enlarged; in that case it is firm, tough, and atrophic, with a large increase of its trabecular tissue and almost entire disappearance of its pulp. Similar changes are to be found in the *liver*, producing large, tough tumors, which, if pigment matter is present in considerable quantity, may be of a chocolate color. In rare cases the liver is contracted, granular, and bearing a certain resemblance to the liver of cirrhosis. Amyloid degeneration is also sometimes present, simultaneously with that of the spleen. In pernicious and severe remittent fever the *kidneys* are sometimes found in a high state of hyperæmia. According to the observations of Key,[1] they are more or less swollen after long-continued intermittent fever; and under the microscope, in addition to amyloid degeneration of the Malpighian corpuscles, and of the various branches of the renal artery, show a marked hypertrophy of the interstitial tissue from the new development of round cells, nuclei, and branched connective-tissue corpuscles. The basement membrane, furthermore, is greatly thickened by several layers of thin, spindle-shaped bodies imbedded in its homogeneous substance. Through the successive transformation into epithelium of one cell layer after another of the basement membrane lining the interior of the tubules, and through degeneration and separation of the same, wax-like cylinders or granular molecular masses are produced, which fill up the urinary tubules. Diffuse inflammation, resulting in the enlarged kidney, may also be observed often enough. I do not venture to say whether the contracted kidney also occurs after intermittent fever. The fact that very considerable accumulations of pigment matter are found in the spleen, the liver, and the kidneys, in chronic infection and malarial cachexia, has already been stated.

---

[1] Canstatt's Jahresber., 1862, IV, p. 50.

In one case, that of a once vigorous man, twenty-eight years of age, who came from a "polder" region, and had suffered from intermittent fever for years, I found, in addition to an enlarged liver and spleen, and dropsical manifestations, a high grade of fatty degeneration of the heart, with numerous points of hemorrhage, mostly of the size of a pin-head, scattered throughout the tissue of the organ, without any pigment accumulations in the blood-vessels.

Analysis of the Individual Symptoms.

It was demonstrated by de Haën, as early as the year 1767, that there was an *elevation* of the *temperature of the body*, even during the stage of chill, a fact which has since been confirmed on every hand. But even some time before the chill, from three-quarters of an hour to several hours, during the prodromal period, the use of the thermometer in the mouth or the axilla shows a distinct elevation of temperature. From this time it rises rapidly, and generally reaches its maximum at the end of the cold stage; sometimes, however, this point is not attained until during the hot stage, or, according to Michael,[1] until the beginning of the stage of sweating. As soon as the maximum is reached, the temperature remains stationary, with small fluctuations, for some time (one to two hours,—in one case of Bärensprung's even four hours), and then begins to fall, at first slowly, afterwards, during the sweating stage, faster, without, however, quite reaching the normal standard at last. The maximum usually runs to a point from 5.5° to 7° over the normal standard, scarcely ever being under 102.2° Fahr., often over 104°, from 106.3° to 106.7°, and in some cases has run as high as 108.5° and 109.4° (Zimmermann). The different types of the disease do not show any difference in the height of the temperature. During the chill, the temperature of the peripheral portions of the body is lowered from 9° to 12.5°, and even more, owing to the contraction of the smaller arteries, the scanty blood supply thus caused, and the consequent diminished tissue metamorphosis; during the hot stage, on the contrary, the heat of the surface of the body is increased. The fall of temperature, with few exceptions, occurs more slowly than the rise, and, indeed, as a rule, by a not quite regular descent. This is true of simple quotidian fever,—in the tertian form it is claimed that the fall is sometimes more rapid than the rise.

During an intermission the temperature falls to the normal standard, or even below, which Griesinger attributes to the anæmia and disturbances of nutrition resulting from the malarial disease; in rare instances, especially in case of certain gastric disturbances, it does not fall quite to the normal standard. The period of time which elapses from the beginning of the rise of temperature to the end of its fall usually averages from nine to eighteen hours in quotidian fevers, and from sixteen to thirty-two hours in tertian, the maximum elevation being usually attained in five or six hours from the beginning of the rise, though it may sometimes be earlier or later.

Under some circumstances the rapidity and uniformity with which the temperature rises may be taken as a criterion of the severity of the attack. In the severer forms of remittent fever, the height to which the mercury rises may, to some degree, be considered as an indication of the amount of danger to life. In this form the temperature usually rises rapidly to its acme during the stage of chill, remains at that point, with insignificant fluctuations, for some time, at the longest for three days, and then, in the event of a favorable course of the disease, diminishes gradually, with morning remissions and evening exacerbations. Barndel[1] has observed that when the temperature remains at its acme for several days, and the remissions are indistinct, we may be prepared for a grave case.

---

[1] De calore corporis humani diss. Lipsiæ, 1855.

Without entering minutely into the theories on fever, I will yet add a few remarks. The doctrine that the elevation of the body-temperature, and the increase of the heat supply in the body, is due to the diminished loss of heat during the cold stage, may be looked upon as exploded, for the temperature is high before the subjective feeling of chill and the peripheral cooling of the body occur; and, even where the temperature does not determine the question of an increased process of oxidation and a greater production of heat, we conclude this to be the case from the increased excretion of urea which often exists for some time before the fever. The production of heat rises till the maximum of temperature has been reached, as is shown by the thermometer, and which usually happens at the end of the cold stage; then it begins to sink gradually, but not at the rate indicated by the thermometer, inasmuch as the giving off of heat and the consequent cooling of the body is increased during the hot stage and the stage of sweating, and, therefore, a record of temperature is given which corresponds as little to the quantity of heat produced as does the record given during the cold stage, when, on the contrary, the excessive contraction of the smaller vessels and anæmia of peripheral parts diminish the giving off of caloric.

In the elevation of temperature that takes place during a paroxysm of fever, however, it is not merely a matter of increased production of heat and diminished loss of heat, but we have to do with a modification or disturbance of the power that *regulates* the heat of the body, by which disturbance, according to Liebermeister, the average grade of temperature is raised to a higher point than is normal. This has given occasion to a belief in the existence of a special centre presiding over the regulation of animal heat. Whether this is an excito-caloric or a moderating centre, or whether it possesses a double character, this much seems to be certain, that in fevers, and especially in intermittent fever, this nerve-centre is so affected by the specific malarial poison that it is disturbed in its functions, either momentarily or for some time, whereby intermissions and remissions are produced. A certain number of the manifestations that accompany the fever, headache, dizziness, malaise, violent trembling and convulsions, etc., are either likewise dependent on the direct influence of the malarial poison on the central nervous system, or must be regarded as results of the increased supply of heat in the body, and as the effect of the heated blood on those nerve regions.

---

[1] Rec. de mém. de médec. milit., 1866, Août, p. 118. Hirsch u. Virchow's Jahresbericht, 1866, p. 178.

In *masked* malarial affections certain nerve-trunks, or at least their roots, seem to be directly attacked in the same way, before the centre that presides over the regulation of heat is involved, inasmuch as such paroxysms are usually not accompanied by a high temperature. (Sensitive, motor, vaso-motor, and trophic nerves are attacked—the two latter in cases of hyperæmia, œdema, and inflammatory disturbances of nutrition.[1]) The exact determination of the amount of urea that is excreted during the attacks, which, so far as I know, has never been accomplished, might perhaps throw a good deal of light on this form of fever. In chronic malarial infection, whether of a primary or secondary nature, we meet with still more complicated relations, which cannot at present be entered into in full.

As was intimated above, the changes in the *blood*, at least so far as we can demonstrate them, are secondary in their nature, and partly the result of a defective development of blood, owing to disease of the stomach and bowels, and the blood-making organs, the spleen and lymphatic glands; partly dependent on the destruction of numerous red blood-corpuscles and the transformation of hæmatine into pigment; partly owing to consumption, caused by the high temperature, or by pernicious complications, or by the loss of albumen through coincident disease of the kidneys, etc. Careful examinations also demonstrate *a diminution in the number of the colored corpuscles, a lessening of the fibrine and albumen, and an accumulation of yellowish-red, brown, or black pigment matter.* It has not yet been possible positively to demonstrate a multiplication of the colorless corpuscles of the blood.

---

[1] In the latter instances, the local disturbances seem to disappear during the intermission, while the paroxysms are short and not too often repeated. In the opposite contingency, the vitality of the affected organs suffers, and the changes, especially œdema and the inflammatory changes, remain during the intermission and increase in proportion to the number of attacks.

In a patient who is still under treatment in my clinic, with primary chronic malarial infection, and very distinctly enlarged liver and spleen, I have been able, as I have in some other cases, to demonstrate the extreme smallness of the colorless corpuscles, which are no larger than the red ones. The diminution of the albuminoid elements and the red corpuscles explains the pale, anæmic, and partly œdematous condition of the skin, the dropsies, the dyspnœa, and palpitation of the heart, the fact of patients being so easily fatigued on the least exertion, and a number of other similar symptoms. Frequent epistaxis and other hemorrhages, the result of the lowered nutrition and tonicity of the walls of blood-vessels, is also remotely attributable to the same cause.

The accumulation of *pigment* in the blood (melanæmia[1]), often very considerable, which can easily be shown in a drop of blood drawn during life, occurs less frequently in simple acute malarial fever than in the tedious, long-continued, or pernicious remittent forms, and in chronic infection and malarial cachexia. As we know very little positively with regard to the method of its production, it will probably be long before we can explain the circumstance that this condition exists in the blood of one individual, after a certain number of attacks of fever, whose number, length, and intensity bear no comparison with those which have been suffered by another similarly constituted person, in whom no pigment can be found; in fact, that in some subjects this condition is not developed even after prolonged illness. A number of serious disturbances are associated with this condition of the blood. Through obstruction of the capillary blood-vessels, or thickening of their walls, we may have ischæmia, collateral hyperæmia, rupture of the vessels, and extravasation of blood, with consequent functional disturbances of the organs in which this occurs. In this way we may explain, amongst other things, the ashy gray color of the skin, an interference with secretion in the liver by obstruction of the branches of the portal vein, and of the flow of bile (icterus[1]), secondary atrophy of the liver followed by ascites, hemorrhage from the stomach or bowels after obliteration of numerous hepatic vessels; furthermore, dangerous brain symptoms, as delirium, coma, convulsions, and sometimes sudden death. It must here be remarked, that while it is true that in many severe affections of the brain immense quantities of pigment are found in the blood, yet this does not justify the conclusion that all brain affections depend on this cause, for in many severe cases of these affections, while pigment is found in the blood, the brain is free from it. At other times the most profound nervous disturbances exist where it is impossible to demonstrate the presence of pigment in the blood, or in any organs of the body, and I have even found pigment in the walls of the cerebral vessels, where no brain symptoms whatever had appeared during life. It is evident, from what has been said above, that the most dangerous nervous symptoms are often accompanied by hyperæmia, œdema, and softening, which may occur without any accumulation of pigment, and which are sufficient to account for all the symptoms. In case of the suppression of urine, we may, according to Griesinger, suspect uræmic intoxication. The algid form of intermittent gives unequivocal evidence, during its course, of gradually increasing paralysis of the heart, which also accounts for most of its symptoms, and which Griesinger thinks may probably be referable to an accumulation of pigment in the muscular tissue of the heart, though the evidence of this is as yet lacking.

---

[1] Compare with Vol. VII of this Cyclopædia, and *Frerichs*, Klinik der Leberkrankheiten, 2 Aufl. Braunschweig, 1861. Bd. I, p. 325.

---

[1] According to *Grohe* (Virchow's Archiv, Bd. XX.), by the accumulation of pigment matter in the smaller biliary ducts.

Pain in the back of the neck, including the first dorsal vertebra, which is so common in ordinary malarial fever, showing itself either spontaneously or on pressure, probably depends on hyperæsthesia of the nerve-roots caused by hyperæmia, and, although occurring most frequently in intermittent fever, cannot be considered as diagnostic of this form of disease.

The changes which take place in the *skin*, as regards color (pale yellow, greenish yellow, earth-colored, or ashy gray) are partly due to anæmia, partly to the accumulation of pigment; and the image of malarial cachexia often stands out so plainly on the countenance that he who has had occasion to see many such patients can often make out a probable diagnosis from the color of the face. In other cases, with gastro-intestinal catarrh and closure of the ductus choledochus, or in pernicious, bilious intermittents and remittents, the skin is more or less sallow up to the point of being bronzed or olive-colored. In malarial cachexia the skin is usually dry and desquamates. After this condition has lasted for some time the subcutaneous adipose tissue is materially diminished. In simple malarial fever, copious warm sweats relieve the patient notably, and are, as a rule, followed by complete apyrexia; profuse cold sweats are an unfavorable prognostic sign.

An eruption of *herpes* is of very frequent occurrence, and may be of value in a diagnostic point of view, especially as between this disease and typhus, as it is rare to meet with it except in malarial fever and pneumonia. The eruption appears on the lips, the nose, and other portions of the face, as well as the gums and the tongue,—seldom on other parts of the body. It is usually an accompaniment of simple malarial fever. It appears either at once, during the cold or hot stage, or after the occurrence of several attacks, or even after these have been cured by medication. As a rule, it appears but once, though occasionally relapses are seen. Sometimes herpes appears as early as the prodromal stage, and then, according to Ornstein, of Greece,[1] is of prognostic significance, inasmuch as whitish-yellow and amber-colored scales and crusts point to a light fever; brown ones indicate active and rapid congestion, and therefore a less favorable course; while painful crusts herald a pernicious attack. The nature of the connection between herpes and intermittent fever is at present unknown. I have sometimes seen herpes occur with great frequency, the vast majority of patients, at some given time, being attacked thereby; according to my observation it is more likely to appear during an epidemic in the spring than in the autumn. Other skin diseases, as roseola, purpura, urticaria, erysipelas, etc., are often encountered; petechiæ appear chiefly in pernicious and severe remittent and continued fevers, as well as in children and in previously feeble persons during simple intermittent. Acne, furuncle, and anthrax are sometimes observed in malarial fever, and especially after its cessation, so that they have been thought by some to have a critical significance; while others endeavor to explain the etiological relation between these skin diseases and intermittent fever. Poor[1] believes that they owe their origin to pigment embolism of the capillaries. Noma, too, is not seldom found in atrophic and weakly children.

---

[1] Revue de Thérap., 1861, Janv. 1.

The *circulatory apparatus*, too, shows abnormal manifestations in intermittent fever. The cooling of the surface of the body which takes place during the stage of chill, depends, to a great degree, on a peripheral arterial cramp or contraction, whereby the pressure on internal organs is materially increased, even sometimes causing rupture of the spleen or heart,[2] as well as capillary hemorrhages in other organs. The increased internal pressure, and the simultaneous lowering of the force of the heart as the result of a lesion of innervation characteristic of the disease, leads to venous congestion (blue color of the lips and fingernails). At the same time the area of dulness over the heart is sometimes enlarged, oftener in width than in length, owing to the over-distention of the right side of the heart. Griesinger observed such a case, in which a very wide heart returned to its normal size again at the end of the illness. Duchek saw a patient in whom, during the attack, the dulness reached as high as the second intercostal space on the left side, and during the intermission fell to the fourth rib again, without at any time giving evidence of an increase in width. The impulse of the heart against the wall of the chest remained at the normal point, forbidding the idea that the heart might have been pushed upward by an enlarged spleen. During the hot stage the cramp of the vessels is relaxed and the previously small and hard pulse becomes fuller; its frequency is always increased in the febrile intermittents, without the rise and fall in its frequency corresponding to the rise and fall of temperature.

---

[1] Prager Vierteljahrschrift, 1863, Bd. I.

[2] *Sebastian*, loc cit., p. 187, says: "I know of a case in which a patient, who exposed himself to cold on the approach of an attack, died from rupture of the right auricle."

It is not uncommon to find systolic murmurs at all the outlets of the heart, or, it may be, only at the origin of the large vessels, as well as in the veins of the neck, and, according to Griesinger, in the region of the spleen. They usually amount merely to arterial murmurs; in anæmic persons they outlast the attack, while in others they are audible only during the paroxysm, and no doubt depend upon abnormal conditions of tension in the walls of the heart and of the blood-vessels. Other murmurs again may depend upon an endocarditis, which is perhaps likewise developed under the influence of malarial poison. In the case of extreme fatty degeneration of the heart already described, I was able, during the whole course of treatment, to perceive strong blowing sounds at both auriculo-ventricular orifices.

The most important complications involving the *respiratory apparatus* were set forth in detail when treating of the general course of pernicious fevers. It is therefore only necessary here to refer to the fact that simple intermittent fever is sometimes accompanied by attacks (either sporadic or to some degree epidemic) of bronchitis, with dry or moist rhonchi, cough, and dyspnœa, which arise during the cold stage, diminish gradually during the stage of sweating, and disappear entirely during the intermission. They undoubtedly depend on hyperæmia of the lungs, with swelling and excessive secretion from the bronchial mucous membrane.

Although, as a general rule, it is true that the *urine* is increased in amount, clear, watery, and of low specific gravity during the cold stage, which is doubtless due to the complete inactivity of the skin caused by the contraction of peripheral arteries; that it is more scanty, red, and of higher specific gravity during the hot stage; and during the stage of sweating is moderately abundant, heavy, loaded with solid ingredients, and throwing down an abundant deposit of urates; yet the exceptions to this rule, dependent on the constitution of the patient, or on some cause connected with the paroxysm, are so frequent, and at times so constant, that one is almost at a loss to determine which is the rule and which the exception. Thus, I have seen perfectly clear, light urine, free from any sediment, in patients who had suffered from repeated attacks of intermittent fever, and whose constitution was weakened thereby, or by other previous illness. The same state of the urine may be observed in cases where the paroxysms are of short duration or irregular in their character, as in the absence of some of the stages; or it may even be found present where none of the above-named conditions exist. The circumstance that the urine is sometimes paler and more abundant during the entire paroxysm than during the intermission (Zimmermann), is attributed by Griesinger to the large amount of water that is drunk.

Allusion has already been made to the fact that the increase in the amount of *urea* excreted furnishes a much more delicate test of the degree of tissue metamorphosis, and the consequent amount of heat produced, than does the thermometer; for even in the prodromal stage, before the thermometer begins to rise, the urine betrays the presence of an increased quantity of urea. This increase rises rapidly in degree until it reaches its maximum, at the end of the cold or the beginning of the hot stage; then, during the hot stage and the stage of sweating, it falls, fast at first and afterwards more slowly.[1] According to Ringer, its increase stands in direct ratio to the elevation of temperature, so that every degree of heat above the normal standard represents a definite quantity of urea. Redtenbacher, in a series of careful experiments which he instituted in the Pfeufer laboratory on ten patients, found that, with the greater amount of urea excreted, the quantity of urine was also increased during the entire paroxysm, and during the intermission both fell decidedly below the normal standard. During the cold and hot stages the amount of urea was three and one-fifth times greater than normal, and during the sweating stage one-third greater than normal; when compared with the quantity excreted during the intermission, it was three and one-half times greater during the cold and hot stages, and one-half greater during the sweating stage; so that during the intermission it stood about one-fifth below the normal standard. The quantity of urine passed was also three and one-fifth times as large as was normal, during the cold and hot stages, and one-third larger than normal during the sweating stage. When the temperature is brought down to the normal standard by the use of quinine, the excretion of urea, as well as of chloride of sodium, still remains increased for a few days; and this is most marked on those days when, but for the treatment, the paroxysms of fever would have recurred.

---

[1] The determination of the amount of urea and of the chlorides excreted during a paroxysm of intermittent fever was made by *Traube* and by *Jochmann*, and the same investigations have of late years been pursued on a larger scale by *Ringer* (Lancet, 1859, Aug. 6th), *Redtenbacher* (Henle-Pfeuf. Zeitschr., III Reih, III, B.), *Hammond* (Gaz. méd. de Paris, 1859, No. 42)—who, it is true, arrives at results quite opposite to the others—and *Uhle* (Wien. Wochenschr., 1859, p. 100).

The amount of *chloride of sodium* excreted by the kidneys is also increased during the paroxysms, although it bears no relation to the temperature, but only to the quantity of the urine. Howitz found it present in normal quantity, Hammond and Uhle found it diminished, and Gieseler likewise found little or no chloride of sodium in the Hannoverian marsh fevers.

No thoroughly satisfactory observations have been made with regard to the proportions of *uric acid* and *phosphoric acid;* according to the scanty data which I possess, it would appear that both are materially diminished on the fever-days.[1]

The following abnormal ingredients of the urine must also be mentioned. It is no very uncommon thing to find *albumen* present in considerable quantities. This may correspond with the hyperæmias of pernicious and remittent fevers, and is then usually accompanied with an abundant admixture of blood; but it appears that even simple malarial fevers may show a highly albuminous condition of the urine, and even renal hemorrhages, during their course, Griesinger having reported such a case, and my own experience, of late, giving confirmation to the same thing. Albumen is to be found in the urine either only on the fever-days, or during the intermission as well (fibrinous tube-casts have also been observed), and disappears on recovery. Still, cases do occur which pass into a chronic diffuse nephritis. Patients often complain of discomfort or severe pain over the region of the kidneys. As a matter of course, disturbances of circulation in the kidneys, with albuminuria and hæmaturia, may arise from the obstruction of vessels by pigment matter. More frequently, however, albumen is to be found after long-continued attacks of intermittent fever, or while a person is laboring under the malarial cachexia, and is then constant and quite abundant, depending on the existence of an enlarged kidney,[1] or on amyloid degeneration of the renal vessels. *Biliary coloring matter* is found in the urine in icterus, as a result of gastro-duodenal catarrh, and in icteric intermittent. *Blood* is present, especially in the above-described remittent and continued "fièvre bilieuse hématurique." Of late years, some isolated reports by Burdel have appeared with regard to the occurrence of *glycosuria* in intermittent fever.[2] In the pigment kidney the so-called fibrinous casts are sometimes found covered with finely granular or flaky pigment.

---

[1] *Nicholson* (Madras Quarterly Journ., July, 1863; Canstatt's Jahresber., 1864, IV., p. 60), could find no uric acid during the paroxysms, and found phosphoric acid reduced to one-eighth of its former amount, whence he concludes that there is depression and palsy of the nervous system. Others, as *Hammond* and *Ranke*, found an increase of uric acid during the attacks.

Dropsy, which constitutes so important a symptom of malarial cachexia, running its course either with or without nephritis, and, in the first case, according to Rosenstein, attaining a higher grade than in nephritis from any other cause, also shows itself, to a greater or less degree, in the masked and in the simple forms of intermittent fever. When it is confined to the lighter forms, and consists only in moderate œdema of the legs and the face, it may be accounted for by the disturbances of circulation that take place during the attacks, the anæmic condition of the patient probably also contributing thereto. Higher grades of the same, which, according to Griesinger, may develop themselves in from two to six days, with a sluggish pulse and great languor, and in which the urine may contain no albumen, or only traces of it, are more difficult to account for. Such cases are not very unfavorable, and may recover within from one to several weeks. In one such case, observed by me, œdema of the legs and the conjunctiva persisted for months, the urine being free from albumen, dark-colored, of high specific gravity, scanty, but without sediment, and the patient having grown anæmic. The heart's action was very weak and the pulse small, and I believe, therefore, that we may safely attribute these cases also to thinning of the blood and disturbances of circulation. Where, in addition to the above causes, we have obstruction to the portal circulation, due to secondary atrophy of the liver, ascites may be developed to a high degree, throwing the dropsy of other parts into the background.

---

[1] *Rosenstein* (Pathologie u. Therap. der Nierenkrankheiten, 2 Aufl. Berlin, 1870, p. 218) found the urine in nephritis, under these circumstances, highly albuminous, and the coagula in the sediment strongly impregnated with urates and free uric acid crystals.

[2] Union méd., 1859, p. 139, and 1871, No. 105.

Various disturbances may be met with on the part of the *intestinal canal.* The nausea and retching so frequently present in the lighter forms of chronic malarial intoxication seem to be nervous symptoms, inasmuch as they often disappear quickly, or even suddenly, to make room for some other symptom. The gastric symptoms that appear during the prodromal stage, or the attack itself, indicate acute gastric catarrh, which, according to Muhry, may result from the direct influence of the poison on the tongue, the mouth, and the gastric mucous membrane. Whether this is the case in intermittent attacks that have a longer period of incubation, I do not pretend to say, but the instances of rapid outbreak of the disease, immediately after infection, seem to prove that the direct influence of the poison on these parts is at least possible. In simple intermittent diarrhœa or constipation may also be present, in addition to the evidences of a mild gastric catarrh. When this gastric catarrh is more severe, and of longer duration, associated with jaundice and with a general febrile condition, the intermittent fever assumes the form of a remittent. More serious disturbances of the gastro-intestinal canal are to be found only in pernicious fevers; such, for instance, as hemorrhages from the stomach or bowels, incessant vomiting, and choleraic or dysenteric stools.

The disturbances of the *liver*, in simple malarial fever, are usually confined to catarrh of the biliary ducts, with jaundice and acute swelling, probably as the result of hyperæmia, which is, however, less frequent than in the spleen, and is usually accompanied with pain in the region of the liver, occurring either spontaneously or on pressure. In such cases Burdel found a pretty abundant admixture of sugar in the urine. The jaundice in icteric pernicious fever and in the fièvre bilieuse hématurique is, in the great majority of cases, to be looked upon as of catarrhal origin, more rarely as depending on an obstruction of the smaller biliary ducts by pigment matter. At the same time, cases have been observed in which the fæces were colored, and in which the contents of the bowels were found strongly tinged with bile, in spite of the most extreme jaundice. Chronic tumors of the liver and amyloid degeneration favor the cachexia; atrophy of this organ leads to stasis in the radicles of the portal circulation, and especially to ascites. In the case alluded to above, with a large tumor of the liver and the spleen, leucine was found in the urine.

Tenderness and actual spontaneous pain in the *spleen*, or pain on taking a deep inspiration, or on coughing, sneezing, moving the body vigorously, or producing pressure over the organ, together with enlargement of the same, are phenomena worthy of attention in malarial fever. Duboué[1] and Derblich,[2] however, are mistaken when they represent the first symptom as being seldom absent, and regard it as of great diagnostic value, inasmuch as in many cases it may be sought for in vain, especially in all those of a chronic character. It arises from the great and rapid stretching of the capsule of the spleen during the paroxysm, and is to be found, above all, in children and young persons during acute attacks, being less noticeable in older persons, with thicker capsules that are not so easily distended. When the spleen has, furthermore, formed adhesions to neighboring organs, tenderness in this region, on moving or shaking the body, may remain persistent.

---

[1] Moniteur de sc. méd., 1861, No. 83 et seq.

[2] Wien. med. Wochenschr., 1864, No. 24.

Enlargement of the spleen, from the most insignificant to the most formidable in degree, is a far more constant condition. If the enlargement is not great, the diagnosis must be made by means of percussion, as the tumor does not extend below the margin of the ribs. In more serious enlargements, however, the organ extends, to a greater or less degree, into the cavity of the abdomen, occupies a more vertical position, and may fill a great portion of the left side of this cavity. These large tumors, on account of their weight, and by reason of an elongation of the suspensory ligament of the spleen, often sink far down towards the pubes, and are then characterized by great mobility. In one of my patients, when lying on the right side, the edge of the tumor extended into the right lower region of the abdomen. Swelling of the spleen is even found in the new-born infants of mothers suffering from malarial fever. In ordinary intermittent fever, the enlargement sometimes takes place as early as during the prodromal stage; usually, however, not until the stages of chill and heat, receding again during the sweat and the period of apyrexia. The spleen may swell rapidly or slowly, and, as a rule, attains a larger size in children and young persons than in those more advanced in years, the capsule of whose spleen is thicker and less yielding. It is also believed that the enlargements are more considerable after quotidian and quartan than after tertian fevers. When the attacks are often repeated, an increase of volume remains after the paroxysm is past; but the very largest tumors of the spleen may also be developed without any actual paroxysms of fever or any demonstrable rise of temperature. In such cases we have to deal with an insidious, hidden process, a chronic infection, in which, according to my opinion, nothing but an accurate determination of the daily excretion of urea can give us any measure of the slow but steady progress of the disease. According to some observations made by me, in such cases, the amount of urea was increased.

The very evident and almost constant way in which the spleen is involved in intermittent fever, suggests the idea that the two are very nearly related. With our present knowledge we can no longer attribute the swelling of the spleen to an overfilling of the organ with blood during a chill alone, as it may attain to the very largest size in chronic infection without chills. Piorry reverses the old proposition, and designates intermittent fever as a result of disease of the spleen; Meckel and Duchek also declare the spleen to be the primary seat of disease. On the other hand, again, according to the reports of Jacquot, Saurier, and others, people are said to die of malarial fever, in Africa, without the slightest swelling of the spleen. A singular case is also narrated,[1] in which a man received an extensive wound of the wall of the abdomen on the left side, through which the spleen escaped; as it could not be returned it had to be removed. The patient recovered from the operation, and had malarial fever afterwards, just the same as before, although eventually a post-mortem examination showed nothing but the shrivelled rudiment of a spleen left. At the same time it should be remembered that certain organs, such as the lymphatic glands, the spinal cord, etc., have been found capable of acting vicariously for the spleen; and it might not be uninteresting, in fatal cases of intermittent fever, with or without enlargement of the spleen, to direct some attention to the state of the spinal cord.

The influence of malarial poison on the spleen may, perhaps, be accounted for on the supposition that, on the one hand, it paralyzes the vaso-motor nerves, or the coats of the vessels in this organ, thus inducing hyperæmia; on the other hand, that it directly injures the blood in the vessels, and in the parenchyma of the gland. By the latter means the red blood-corpuscles are disintegrated and transformed into pigment, and the white corpuscles are hindered in their development, which accounts for the small, rudimentary specimens of the same found by me. In case of the long duration or frequent repetition of the hyperæmia, abundant proliferation of the adenoid connective tissue and permanent enlargement result. Perhaps the spleen may also be regarded as a depôt of deposit for the malarial poison.

The *blood* is only a vehicle for the poison, which, by hyperæmia and destruction of blood corpuscles in such organs as are disposed thereto, may occasion, at such points, the local development of pigment-matter. The *nervous symptoms* are merely expressions of the disease, which, it is true, play a very prominent rôle in malarial fevers, and depend upon irritation of the most varied nerve-tracts through the malarial poison, or through the blood altered by this poison, as may be seen most plainly in the masked fevers. The effect seems to be produced especially on the brain and spinal cord,[1] perhaps also on the roots of individual nerves.

---

[1] *Canstatt*, Jahresbericht, 1860, IV, p. 70.

The question of *the cause of the rhythmical recurrence of the paroxysms* remains, as yet, unsolved. This periodicity used to be attributed to some influence of the moon, and was considered as a manifestation of increased vitality dependent upon that luminary. Again, it has been explained on the ground of a tendency in the nervous system to work by rhythmical impulses, or by supposing that the poisonous material had the power of appearing in the blood periodically, and of manifesting itself, sometimes in a longer, sometimes in a shorter time. Others have attributed it to a natural tendency in the healthy subject to daily fluctuations of the pulse and temperature. Heschl explains the periodicity of malarial fever depending on the disintegration of red blood-corpuscles, on the theory that the production of these corpuscles is not a constant, uniform process, but takes place by starts, with a daily maximum and minimum.

It is a striking fact that the attacks of fever most frequently occur between midnight and noon, at a time, therefore, when the organism has gone the longest without food, and when physiological waste, as well as a pathological waste caused by the destruction of numerous corpuscles, call most loudly for repair. We might, indeed, be tempted by this circumstance to suspect some connection between the paroxysm and the principal meal-times. This view is further sustained by the fact that the attack may be diminished in intensity, and when it is light—especially in the case of masked fevers and intermittent neuralgia—may even be prevented, by the use of means that lessen tissue metamorphosis, and therefore physiological waste, such as strong coffee and rum (a popular remedy), or other spirituous drinks. Ritter speaks of the admirable results sometimes obtained from black coffee, even in cases where arsenic failed, and quinine did not always answer his expectations. May not this hypothesis account for the popular treatment, known in many lands, of attempting to break up the fever by hearty eating?—whereby, in reality, the paroxysms do sometimes disappear. It is true that Griesinger and Duchek have thus far failed in producing any effect on the recurrence of the paroxysms by alterations in the hours of meals and other relations of life; nevertheless, in my opinion, this question should not be altogether ignored in the future.

---

[1] *Griesinger* (Infectionskrankheiten, Edit. II, p. 40) narrates some cases that bear upon this. In one patient with complete paralysis of sensation and motion of the lower part of the body, due to fracture of the tenth dorsal vertebra, the paralyzed portions remained unaffected, while the rest of the body underwent a complete paroxysm of fever, with chill, heat, and sweating. In another patient, after the cure of malarial fever by quinine, regular paroxysms of horripilation and cold continued in an arm which was the seat of a suppurating wound. In a woman sixty-two years old, observed by Griesinger himself, who had suffered for four years from traumatic anæsthesia of the left hand, light attacks of intermittent declared themselves by chilliness and pain in that arm. I myself treated a tobacconist in whom malarial fever showed itself by severe, regularly intermitting neuralgia of both lower extremities, with chill and subsequent heat in the same.

Course and Sequelæ of the Disease.

Simple malarial fevers run their course in accordance with the types indicated above, one type occasionally running into another. Recovery may undoubtedly take place without the assistance of medicine, if the patient withdraws from malarial influences by taking up his abode in some non-malarious region; or if, as is the case in some places, malaria is endemic only at certain seasons of the year, and then disappears. Often, however, this recovery is only apparent. The paroxysms, it is true, become more indistinct and weaker, and finally cease altogether; the patient declares himself free from fever, and the physician can only recognize the remaining, almost unnoticeable exacerbations of the same, by the most careful use of the thermometer. At the same time the patients grow more emaciated, weaker, and more anæmic; œdema of the lower extremities supervenes, the skin assumes a cachectic hue, and the liver and spleen are more or less enlarged. If this condition is recognized and treated early enough, especially if the patient is removed from the malarious region, recovery is still possible. Usually, however, the cachexia advances in its course unimpeded, or interrupted, now and then, by a more decided paroxysm of fever, and the patient eventually dies of marasmus or of one of the sequels of malarial disease, such as amyloid degeneration of the liver or spleen, cirrhosis of the liver, with its accompaniments, Bright's disease, scurvy and tuberculosis, or chronic diarrhœa. In rarer instances death follows suddenly by apoplexy—perhaps the result of pigment embolism of the brain—or by pneumonia, or some other acute disease. The cachectic condition is especially liable to be developed during the tender years of childhood, and then usually carries off its victim.

The simple malarial fevers of our part of the world present no special difficulty in the way of treatment, provided this is undertaken while they are in the acute stage. But the cessation of the attacks for a time is no evidence that the disease is eradicated. *Relapses* are very liable to occur after a variable period, which, being overcome by appropriate treatment, may, after a time, again occur, and continue to do so three or four times.[1] The possible doubt as to whether these are true relapses may be answered by the fact of the great regularity with which they are liable to recur at the end of seven, fourteen, twenty-one, or twenty-eight days from the last paroxysm, or on days on which, if the fever had continued, a paroxysm would have fallen. The very brief pause, often of but a few days, between the first attack and the relapse, as well as the fact that in some instances the patients have withdrawn from the malarious region immediately after the first attack, all argue against the idea of a new infection, and in favor of the theory of the relapse of a disease not yet extinguished. Relapses are to be met with not only in simple malarial fevers, in the masked, pernicious, and remittent forms, but, even in those suffering from malarial cachexia, paroxysms of fever often show themselves, which still further undermine the constitution of the patient. The type of the disease may also be changed in relapses; indeed there seems to be a tendency to assume the shorter types. Thus Griesinger reports 64.5 per cent. of tertian and 29.2 per cent. of quotidian fevers in a certain lot; afterwards, during the relapses in this lot, only 35.4 per cent. of tertian and 51.6 per cent. of quotidian. Aside from certain injuries and bad conditions that were mentioned under the head of predisposing causes, age seems to have the greatest influence in determining the occurrence of relapses. Griesinger found during the ages of from one to ten years 64 per cent.; from ten to twenty years 52 per cent.; while from twenty to ninety there were but 38 per cent. of relapses. The period of time within which they may show themselves is very variable. As a rule, they occur within a few weeks, and we do not consider the malarial influence as overcome until some six or eight weeks have elapsed without an attack. Individual instances occur in which relapses do not appear until after some months.[1] Assertions such as those made by Todd and Guetano, and illustrated by cases, to the effect that the marsh-poison, having once penetrated into the system, can never be entirely driven out, have thus far met with very little support.[2]

---

[1] *Griesinger* (Infectionskrankheiten, II Aufl., p. 43), at the Tübingen Clinic, saw 208 relapses in 414 cases (50 per cent.); of these 208, a second relapse was observed in 113 (54 per cent.); 50 of the 113 had the fever a third time (40 per cent.); and 17 out of the 50 (34 per cent.) relapsed for the fourth time.

The *sequelæ* of this disease entitled to mention, aside from chronic secondary malarial cachexia, are, diffuse nephritis in the form of the enlarged kidney, amyloid degeneration of the kidney, the same of the liver and spleen, contractions of the liver, the hemorrhagic diathesis, scurvy, tuberculosis, certain nervous affections, such as neuralgia, conditions of paresis and paralysis, as well as various psychical disturbances which supervene after severe intermittent fevers, and are perhaps due, as some believe, to pigment obstructions. Here, according to Griesinger,[1] instead of the ordinary paroxysms of chill and heat, we find intermittent paroxysms of insanity, which, with the cessation of the periodicity, assume the remittent or continued type, and may pass into chronic mental diseases. More frequently, however, psychical disturbances appear during the period of convalescence, as sequels to the disease, or not till months after it has been subdued, particularly following long-continued, severe attacks of fever, especially of the quartan type. After severe epidemics, *abortions*, or premature labors are frequent, as is declared by Sebastian and others, and confirmed by the statistics of the lying-in asylum at this place. Ritter, on the other hand, found neither abortions nor any deviations from the ordinary course of pregnancy and parturition.

---

[1] Two cases by *Braune*, Archiv f. Heilkunde, XI Jahr., p. 70.

[2] *Ritter* (Virchow's Archiv, Bd. 50, p. 164) speaks of what he calls "after epidemics," as, in his experience, the year after the prevalence of an epidemic of acute malarial infection there is an epidemic of chronic infection (1827 and 1869). He explains this, as a general rule, as follows, that in addition to an acute infection a certain number of chronic infections take place, which remain latent during the winter and are called into activity by various circumstances during the spring. He thinks that the two epidemics are proved to belong together by the fact of their occurring within the same area and affecting the same individuals—often invading the same houses and even attacking the same persons. Probably the actual condition of things is this: that some of these persons are laboring under primary chronic infection, others under secondary malarial cachexia, perhaps in both cases with feeble relapses of the same.

Prognosis.

The *simple*, acute intermittent fevers of our part of the world, under systematic treatment, warrant a favorable prognosis, as a rule. This, at least, is true, where the epidemic is not severe, or where the circumstances of the patient are such as not perpetually to expose him to the influence of intense poison, nor constantly to renew the disease when it is nearly subdued. The prospect is more unfavorable in the virulent malarious regions of warmer climates, or in the temperate regions during an especially hot year. Furthermore, the individuality[1] of the patient, the age of early childhood[2] or of advanced senility, or a previously weakened constitution may, with a moderately severe acute attack, or the long continuation of single stages, or paroxysms with short intermissions, make the prognosis unfavorable. The most favorable results are obtained in the tertian type, less so in the quotidian, which weakens the constitution of the patient far more, and least so in the quartan form, which but rarely prevails over any considerable extent of country (Berlin epidemic of Wolff). The anticipating of the paroxysms is looked upon as an unfavorable sign, especially if the later attacks increase in severity; it is favorable only when they become lighter and shorter. Postponing of the paroxysms is desirable in the quotidian form, inasmuch as it indicates transition to the more tractable tertian, when the attacks diminish in length and severity; it is unfavorable, however, when they augment in intensity, or when the constitution of the patient is evidently breaking down, as we then fear a change to the more malignant quartan type. The same thing is true of subintrant fever, as constituting a transition stage to the remittent and continued fevers, which we heartily deprecate. The progressive weakening of the paroxysms, while the spleen and liver remain large or increase in size, and while anæmia gradually develops itself, is very unfavorable, as it indicates the certain development of malarial cachexia. Complications affecting any important organ, or the occurrence of any of the sequelæ of the disease, vitiate the prognosis.

---

[1] Pathologie und Therapie der psychisch. Krankheiten, 2 Aufl. Stuttgart, 1867, p. 188.

[2] In Algiers, according to the reports of *Semanas*, there is great mortality among the children of immigrants, in whom malarial fever often ends fatally at the end of twelve to forty-eight hours, with adynamic, bronchitic, or laryngitic symptoms (*Gerhardt*, Kinderkrankheiten, 1861, p. 120).

Masked fevers give a favorable promise, as they generally yield easily to remedies, and probably never, alone, lead to cachexia.

Pernicious fevers, in general, are unfavorable enough, inasmuch as, where prompt assistance fails, they are apt to terminate fatally in the second or third attack; when recovery ensues, they drag through a weary convalescence, or may even leave disturbances of various kinds behind them for a time or permanently. Much depends, in these cases, on the intensity of the epidemic, on the question whether it is advancing, receding, or at its height; on the age and constitution of the patient; the question of the presence or absence of complications, the longer or shorter rhythm of the attack, and whether the intermissions are complete or not, as well as on the form and grade of the disease. Beyond doubt, the most favorable cases are those of the tertian type, with as complete an intermission as possible. The most fatal seem to be the algid-choleraic and the tetanic forms; the delirious and comatose varieties being less so; and those with localization of the disease in the lungs more favorable still.

In the lightest forms of *remittent* and *continued* malarial fever, the prognosis is good; it is doubtful in the medium and worst forms, especially with a persistent low temperature and imperfect febrile reaction, or none at all. According to Veillard, the "fièvre bilieuse hématurique" belongs to the severest forms, and in weakly patients, or when early and effective treatment has been neglected, usually ends in death. We must always look with suspicion on very intense icterus, continuous fever, or very short and incomplete remissions, frequent and violent vomiting, suppression of urine, a considerable admixture of blood with the urine, as well as other hemorrhages and hiccough.

The prognosis in *chronic malarial intoxication* depends upon the grade of the disease, that is, the severity of its manifestations. The size and consistency of the tumors to be found in the spleen and liver give some indication of the gravity of the condition. When the symptoms are moderate, the enlargements inconsiderable and not firm or hard, and when neither ascites nor any of the more serious sequelæ have appeared, the prospect is not so unfavorable. In general, it may be said that primary infection or cachexia, if moderate, offers better chances for recovery than the secondary form, in which the patient is usually reduced in health and constitution by the previous paroxysms of fever. Large tumors of the liver and spleen cannot usually be reduced; still, if the liver is healthy, and the patient removes from a malarious neighborhood, such tumors of the spleen may often be carried for a long time without detriment to the individual.

Diagnosis.

A well-marked attack of intermittent fever is so characteristic that it can hardly be confounded with anything else. When the paroxysms are not distinctly marked, however, and symptoms of gastric disturbance are prominent, one may be led to suspect gastric fever. Diarrhœa and meteorism, if present, may suggest typhoid fever. But the occurrence of herpes, the absence of roseola, and above all the thermometric record, which usually shows morning exacerbations instead of the evening rise of typhoid fever, as well as the non-febrile intervals present, and the well-known epidemic influences under which the patient is laboring, are all important points in the case. As soon as distinctly marked paroxysms, with their characteristic stages and complete intermissions appear, all doubt is at an end. The differential diagnosis between intermittent fever and *pyœmia* is more difficult, as the paroxysms and the high temperature are common to both. Besides the local troubles liable to exist in pyæmia (wounds, the puerperal state, etc.), it will be observed that in the first pyæmic attack the temperature rises much more tardily, and generally falls more rapidly than it has risen ; at the same time it more rarely reaches the normal standard, but is arrested at about 100.5° Fahr. to 101.25° Fahr., and, as soon as the minimum is reached, begins to rise again, without, however, reaching its former height. It is rare to find a complete apyrexia in pyæmia, or an approximatively normal temperature lasting from twelve to twenty-four hours; still, cases do occur in which the temperature falls below the normal standard, to 95° Fahr. or 93.25° Fahr., the pulse usually remaining rapid. Usually the attacks are irregular, and treatment with quinine proves unavailing. We are saved from confounding malarial fever with the *hectic* of tuberculosis, by the absence of those signs characteristic of the latter, viz., local pulmonary affections, emaciation, night-sweats, diarrhœa, etc.

The diagnosis of *masked* intermittents rests on the prevalence of malaria, the well-marked rhythm of the attacks, the sudden appearance and disappearance of the symptoms, with frank intermissions, the occasional febrile manifestations that supervene, such as chilliness, uneasiness, cold or hot sweats, and on the results of treatment.

The *pernicious* fevers, with short intermissions or none at all, are often, and very unfortunately for the patients, mistaken for other diseases, especially when the local symptoms are prominent. Thus, every once in a while I have a patient with comatose intermittent fever sent into hospital with the diagnosis of apoplexy. Not only the outspoken disease, but certain unusual appearances that may be seen in the paroxysms of simple malarial fever that precede the pernicious attacks, and during the intermissions of such paroxysms, are worthy of note, inasmuch as they indicate the danger of a pernicious attack. Such are, severe headache, extreme prostration, an irregular, small pulse, unusual drowsiness, or restlessness and vigilance, apathy, a diminished secretion of urine, as well as an increase in the severity of successive paroxysms. In determining whether any given case is one of pernicious fever, we must consider the question of the endemic or epidemic influences prevalent, inquire into the existence of previous attacks of malarial fever, observe the rhythm of the attack, notice the individual stages, which are usually recognizable, the disappearance or diminution of the symptoms during the intermission and their subsequent aggravation, etc., etc. Where the diagnosis cannot be made with certainty, it is of the utmost consequence that we do not await another attack for the purpose of determining the question; but, in view of the imminence of the danger, that we treat it as a pernicious attack, and at once institute vigorous measures. The choleraic and algid forms are to be distinguished from cholera by the elevated temperature; the algid form is further distinguished by the fact that the stools, if present, are not profuse, and do not appear until late in the disease; finally, according to Rigler, the urine of cholera contains albumen, while that of intermittent does not. The difference between pneumonic intermittent and genuine croupous pneumonia is, that in the former each paroxysm is ushered in by a more or less-marked chill, and that the temperature returns to its normal standard during the intermission, while the objective symptoms, especially the physical signs, abate during this period, to be aggravated again during the subsequent attack. The signs given by Faymoreau for a differential diagnosis between icteric pernicious fever and the fièvre bilieuse hématurique (such as vesical tenesmus and pain in the region of the liver in icteric fever; pain in the region of the stomach in the other form; the absence of blood and hæmatine in the urine, of intestinal hemorrhage, of purpura and of ecchymoses of the skin in the icteric form) do not appear to me to be sufficient. I believe, therefore, that they represent but the one form of disease, which at one time runs a more paroxysmal, at another time a more remittent or continued course, with less strongly marked bilious symptoms. This form is distinguished from yellow fever by the fact that where it is endemic it hardly ever attacks new-comers, as yellow fever does, but persons who have lived in the place for a longer time, at least a year, and who are disposed to intermittent fever. Furthermore, the fièvre bilieuse hémat. has an adynamic character from the beginning; yellow fever, on the contrary, an erethetic character (redness of the face, a glistening eye, injected conjunctiva, hot skin, and strong pulse); in the former, jaundice is one of the earliest symptoms, and hæmaturia a pathognomonic sign, while in yellow fever hæmaturia often does not exist, and jaundice belongs to the later stages of the disease. Finally, in the one case quinine shows its greatest efficacy, in the other it is useless.

TREATMENT.

Malarial fever is one of the diseases in which *prophylactic measures* are of the greatest importance, belonging in part to the domain of public hygiene, and in part falling within the scope of individual effort. Swamps in malarious regions should be laid dry, or, where this cannot be done, care should be taken that the ground is always covered with a moderate sheet of water. Ditches of stagnant water, in the vicinity of towns, should be emptied and kept dry. In marshy bottom-lands such drainage should be established as will regularly and sufficiently carry off the water, and where such lands lie in the neighborhood of rivers, inundations should be guarded against by the construction of dykes. Large tracts of uncultivated land should be brought under careful cultivation; the streets of cities should be paved and kept clean. Furthermore, it is the duty of those having charge of the public health to provide a supply of good drinking-water[1] and to establish poor-houses or places of refuge for the shelterless. The principal prophylactic measures that can be adopted by individuals are, for strangers who are about to migrate to a malarious region, to select, if possible, that season of the year when malarial fever is least prevalent and severe, to avoid those places which are known as special hotbeds of the disease, and to adapt themselves, as soon as possible, to the ways of living of the natives. These precautions are particularly important fo those coming into the tropics from a cooler climate. Here, th winter months—from November to January, for instance, in th East Indies, and from January to March, in the West Indies—ar the most favorable ones in which to arrive, in the hope of becoming so far acclimatized during this period as to be to some degre steeled against the malarial miasms of the succeeding months Even during the passage from Europe, if the sea-voyage is not s long as to give one a chance to become accustomed to a warme climate, certain precautions may be taken on board ship that wil lessen the tendency to fevers. These especially embrace provisions which insure an abundant supply of fresh, pure air, coo sleeping places, cool drink, a light, digestible diet, and the avoidance of all excesses, especially in eating and drinking, the use o alcoholics, etc. On arriving, a somewhat elevated, hilly regio is to be selected, or, where this is impossible, it is better to spen the first few days on board ship, on the high seas, than to b delayed on a marshy coast. All night-work in tropical swam lands is to be avoided; and when one is obliged to travel throug unwholesome regions, the hours of from three to six o'clock i the afternoon are to be selected for this purpose, as at that tim the greatest heat and the most abundant exhalation are past, an the miasms lie in the upper strata of air.

---

[1] In the supply pipes of the houses of Amsterdam may be found water that has filtered through the sandy soil at some hours' distance from the city.

In our climate, too, it is desirable to select the highest an driest residences, far removed from swamps, and never to hav one's sleeping rooms on the ground floor, but upstairs, and, i possible, on the east or south side of the house. Fresh air an cleanliness must be secured, but the bedroom windows shoul always be closed during the night. The clothing must b appropriate to the climate; in warm countries, cotton unde clothing and hose are enough, in cooler climates they should b of wool. One should avoid getting wet, and every means o taking cold, as well as remaining long in the vicinity of swamps, sitting out-of-doors in gardens that lie low, or are enclosed by high trees or walls, especially after rainy weather, and during the damp evenings one should be in-doors. Sleeping out-doors is very injurious, as well as bathing in rivers or lakes after sundown. In tropical regions strangers must also avoid exposure to the powerful heat of the sun, and all undue bodily or mental exertion.

All excesses, sexual as well as dietetic, are to be shunned. The food should be simple and nourishing, and in accordance with the customs of the country, such as highly seasoned food; fresh, good, and not too fat meat; easily digestible fish; fresh vegetables; good, well-baked bread or rice; and for drink, good water which has no possible connection with the swamps. Where such water cannot be had, let it be boiled, or filtered through charcoal, or let the artificial carbonic-acid water be used. Tea and coffee, good wine, and even small quantities of brandy are desirable. Raw salad, watery fruits in large quantities, as well as much milk and beer, are injurious. Every feeling of sickness, and especially any gastric or intestinal catarrh, should be attended to.

The *prophylactic action of quinine*, which was first observed by Bryson in the fevers prevalent on the coast of Africa, has of late received ample confirmation on the part of various writers (Balfour, Baikee, Saussure, and Rogers). The dose used was two and one-half to three or four and one-half grains, two or three times a day. Jilek[1] reports from Pola, that in a garrison of 736 men, 500 of them were given daily, at first three grains, afterwards one and one-half grains of quinine apiece, and a pint of wine, while the remaining 236 men were left entirely without treatment. Ninety-one of the first lot (eighteen per cent.) were taken sick, though in a mild degree and without many relapses; of the last lot, or those without treatment, sixty-eight were taken sick, being twenty-eight per cent. The experiments made by Baxa, in Pola, did not yield the desired result, and he, therefore, recommends, instead of the daily smaller doses, to give a weekly dose of twelve grains. Colin's experience in Italy and Algiers also fails to confirm the favorable influence of this treatment, especially in preventing relapses, and he even believes that the system becomes accustomed to the remedy, and that so it loses its effect entirely. My own observations on this point, made on myself and on others, leave no doubt as to the efficacy of this daily preventive dose. In some instances the attacks are entirely kept off during this treatment, in others they occur only in the lightest forms. I have never seen any protection afforded against relapses, and I have noticed that the system established a tolerance of the remedy, so that, in order to obtain its full effects, one was obliged to omit the treatment for two or three weeks at a time. I have no experience at all in the use of the larger weekly doses. In the more serious epidemics it may, therefore, be recommended to those who are unable to leave malarious regions (which is unquestionably the best prophylactic), that they make use of daily doses of quinine. I recommend from four to five grains morning and night. In the ordinary endemic diseases it is well to pay attention to the first signs of being sick, and a dose of nine to twelve grains of quinine on going to bed at night, for three or four days in succession, is usually enough to break up an intermittent [1]. I have never seen any special results from the use of the tincture of gentian or the tincture of nux vomica, which are also recommended as prophylactics.

---

[1] Wochenbl. der Gesellschaft der wien. Aerzte, 1870, No. 17.

The *febrile attack*, itself, if it is a simple intermittent, usually demands no special treatment in our part of the world. All that is necessary is to keep the patient from injurious influences; but in order to accomplish that, he should, even in the lightest cases, take to his bed on the first appearance of a paroxysm, and abstain from all food until it is over, for fear of inducing vomiting. Only in case of particularly distressing or dangerous symptoms is interference demanded. During the *chill* it is best not to pile on too heavy bed-clothing, as it only fatigues the patient, and to avoid the administration of copious hot drinks, whereby it is vainly hoped that the attack may be cut short. The sensation of chilliness is not removed by such means, and some of those which have been recommended for this purpose, such as warm or steam baths, alcoholic drinks, running or other severe bodily exercise,[1] can only do harm. This sort of treatment is not to be recommended, even in severe and long-continued chills; but we must content ourselves with friction of the skin, warming of the bed, and, if thirst is excessive, with the administration of a moderate amount of warm tea or sugar-water. In persistent vomiting, effervescing mixtures or small doses of opium are indicated. Where, as is most likely to occur in children and aged persons, the vital powers sink, the pulse becomes small and irregular, the respiration shallow, and collapse is threatened, the analeptics may be resorted to with confidence; such as strong wine, ether, camphor, and black coffee internally, inhalations of ammonia, sinapisms and frictions with spirits and mustard, as well as stimulating enemata.

---

[1] The fear expressed by *Neuhold* (Oester. Zeitschr. f. prakt. Heilkunde, 1862, No 18), that the early use of quinine might stimulate the development of dangerous sequels such as dropsy, etc., is entirely without foundation.

During the *hot stage* cool surroundings and light covering are desirable, a moderate indulgence in cool, refreshing drinks, and, in case of vomiting, broken ice by the stomach. If severe headache and delirium are present, giving evidence of considerable congestion of the brain, cold water or ice-compresses should be used, sponging of the body with vinegar and water, and perhaps vinegar clysters. The local abstraction of blood from the temples is only to be practised in robust individuals, and even here venesection[2] must be resorted to with the utmost caution, as it is often followed by alarming collapse. The *sweating stage* requires no particular attention, and above all no measures to force sweating. At the close of this the patient may change his linen, leave his bed, and, if the season of the year is propitious, may go out, after a few hours, observing every precaution against taking cold. If the *apyrexia* is incomplete, he should remain in bed. As regards diet, light but supporting food should be indulged in on the days of the intermissions; on the days when an attack is expected but little should be eaten, as a full stomach readily induces vomiting.

---

[1] *Berndt* (Fieberlehre. Leipzig, 1830, I, p. 629), asserts, it is true, that he has often seen strong persons overcome the chill by hard running, and thus shorten the paroxysm.

[2] The application of wet cups along the spine, as recommended by *Mons* (1836), and by means of which *Zimmermann* claims to have cured forty-one patients out of fifty-two, has met with but little support. If, as has been reported, it is possible to break up the fever during the stage of chill, by *energetic* venesection, so that the remaining stages are not developed, still such a result is rare, and the danger of the severe brain disturbances that may follow it, such as convulsions and coma, is not to be underestimated. *Groh*, of Olmütz, has seen the chill give way, during the cold stage, on the inhalation of cinchonic ether (℈j). The attack was reduced to a minimum, and no relapses occurred. The same effect is claimed for chloroform inhalations.

In *pernicious* intermittent fevers, the expectant plan, which is appropriate during the attack in the simple form, is utterly out of place. Here alarming symptoms crowd on one another, and it is often only by prompt and vigorous measures, on the part of the physician, that the patient can be rescued. And it is not only the alarming symptoms that occur during the attack, but the disease itself that must be combated by specific means, the value of which has been thoroughly established by experience.

Where the fevers have a more sthenic character, and particularly in the pernicious icteric intermittent, it has often proved of advantage to precede or accompany the specific quinine treatment with vigorous purgation. The most appropriate article to use is calomel in large doses, five to eight grains, repeatedly. In the delirious form we should avoid venesection, even in full-blooded persons. Where the abstraction of blood is needed, it should be done by local means; usually, however, cold water or ice-compresses to the head, derivation by enemata, and counter-irritation to the extremities are to be preferred. In coma and apparent death, stimulating enemata, strong irritants to the skin, and tincture of musk subcutaneously may be used; and in violent vomiting, opium, effervescing draughts, ice-pills, camphor, and strong irritants over the region of the stomach. In severe chills, strong friction of the skin and warming of the bed with hot stones; in the algid form, rubbing the skin with spirits of camphor and mustard,[1] or with ice, and internally stimulants, such as ether, champagne, and strong wine, are to be employed. In all cerebral symptoms which are ushered in by great excitement and restlessness, opium or morphine is indicated, and may best be combined with the quinine. In the diaphoretic fevers, the mineral acids, and drinks containing these acids, seem to limit the excessive sweating.

---

[1] Oil of mustard one part, alcohol fifty parts.—*German Ph.*

As soon as the diagnosis of intermittent fever is established we should not delay therapeutic interference, though in the simple form we may await an intermission, for the attack which is already begun is not likely to be arrested nor shortened by the use of the ordinary empirical means that cure malarial fever. The former method, which was to await several attacks, and if there were gastric disturbances, first to remove them by the use of emetics, has gradually been abandoned, inasmuch as it only causes loss of time, and the gastric troubles usually recede anyhow under the use of quinine. In a very few cases, where a fit of indigestion seems to have been the determining point for the attack, an emetic may be advisable. The same rule holds good with regard to laxatives. They are not called for in simple fevers unless there is decided constipation.

A great number of remedies have been recommended at various times, for regular intermittent fever, as well as for the pernicious fevers, the efficacy of which has afterwards been in part confirmed and in part thrown into doubt, and they have therefore soon fallen into oblivion.

Quinine still occupies the front rank among these remedies, the form ordinarily used being the *simple sulphate*, which is given in solution, in powder, or in pills. The former practice of prescribing it in frequent small doses, two grains every two hours, which is even now often met with, ought to be given up, for intermittent fever; because, on the one hand, the entire intermission is often occupied in this way without the patient's receiving a sufficient amount of quinine, and, on the other, the doses given shortly before the attack have no time to act, and an absolutely larger amount of the drug is required to prevent an attack, if given in this way. It often happens that these frequent, small doses will not cure the fever, but merely make the paroxysms shorter and milder. I have many times had occasion to see patients who had been treated in this way for months without effect, and in whom a few large doses broke up the fever at once. The method of treatment recommended by Maillot,[1] and afterwards by Pfeufer, in which one or two large doses are to be given during the intermission, deserves the most unqualified preference. In simple intermittent, 9, 12, 15, or 22 grains should be given as a single dose, or 7½ to 12 grains twice to three times, but in such manner that the last dose shall come five or six hours before the expected attack. In younger persons, from sixteen to twenty years old, one-third less may be taken; children from ten to fifteen years of age should have one dose of 6¾ grains, or two doses of 4½ grains; children from three to six years of age, one dose of 4½ grains, or two doses of 1¾ grains. These amounts are calculated completely to subdue the disease, if it is simple intermittent fever, especially in recent cases. Often, however, particularly in cases that are not very recent, the next, or perhaps even a second attack may occur, although much weaker, and, as a rule, postponed. If *relapses* take place, one dose is usually enough for their removal; where, however, they are frequently repeated, and especially where the patient's constitution is running down, it is well to put him on the more prolonged use of the remedy, combined with iron, if he be an anæmic subject.[1] To adults I usually give 4½ to 6 grains morning and evening, to children 2¼ grains. It is desirable for the sake of avoiding relapses, in malarious regions, to pay special attention to those days on which, in case of the continuance of the fever, an attack would have occurred, viz., in the tertian type, on the third, fifth, seventh, and ninth days, etc., and to give a dose of quinine on those days. It is wrong to suppose that attacks always recur on the seventh, fourteenth, twenty-first, and twenty-eighth days, and that the patient should therefore take a dose of medicine on the day preceding those days. Attacks are very likely to occur sooner and to surprise the patient. The use of cinchona, of the wine, tincture, and extract of cinchona are to be recommended, although less reliable than quinine; the tincture of quinoidine is also a desirable preparation, especially on account of its cheapness. It is well to impress on patients the importance of noting the first warnings of an attack, and as soon as such are observed, let them take a good-sized dose of quinine for several days in succession.

---

[1] Traité de fièvre intermit. Paris, 1836, p. 362.

---

[1] *van Dommelen* (Aerztl. Intelligenzbl., 1864, Beil. 11) recommends pills of sulphat of cinchonia and saccharated carbonate of iron, each, 30 grains, a day, divided int hourly doses. After two or three days the dose is reduced to 15 grains, and finally to 9

In pernicious malarial fever, it is usually necessary, on account of the more imminent danger, to increase the doses considerably, and, as has already been stated, not to wait for an intermission, but to give the medicine during the attack. I give 30 to 45 grains a day, usually in two or three 15-grain doses. Maillot once ordered 180 grains during the twenty-four hours, with a good result. The use of quinine must here be continued until the alarming symptoms have been relieved. When large doses of quinine in solution are vomited, or when there is extreme irritability of the stomach, the extract of opium, or tincture of opium, with aromatics, should be added to the quinine. When even this is not borne, or when there is severe hemorrhage from the stomach, or if the patient is suffering from uncontrollable vomiting, or cannot swallow, quinine must be given by the rectum, in which case it is absorbed with equal promptness. When used in this way the dose is 3, 7½, 9, or 15 grains, and in pernicious fevers as high as 30 grains, combined with some opium. Under similar circumstances, where quinine cannot be introduced into the stomach, it may be given by hypodermic injection, by which method the effect of the drug is produced much more rapidly, and with the use of a much smaller amount. Only about one-third or one-fourth of the dose is to be used hypodermically that would be given by the stomach, 2½ to 3 grains being usually sufficient to overcome a simple intermittent attack. Some claim to have observed (Moore) that if the injection is given during the attack, the latter will be made milder or even cut short. The solution ordinarily used contains one gramme (15 grains) of quinine to two and a half fluid drachms of water, and a few drops of sulphuric acid. Of this from 5 to 15 minims are used as an injection, containing from ½ grain to 1½ grains of quinine. As considerable irritation of the skin, with the formation of abscesses, is liable to occur, Barnatzik recommends the following formula as being less irritating to the tissues: Sulphate of quinia, 150 grains; hydrochloric acid, 50 minims; and distilled water, 4½ fluid drachms. The indications for the hypodermic use of quinine, then, are gastric disturbances; pernicious attacks—on account of the more rapid action of the drug, and in case the patient cannot swallow—larger doses being required in the pernicious than in the simple form; as well as the intermittent fevers of children, particularly if associated with eclampsia. The applicability of this method to the last class is somewhat limited by the tenderness of the skin in children, which often reacts severely under injections.

Quinine has also been used externally by inunction (22 to 30 grains in ℈iv to ℥iijss of lard), or as a spirituous solution, applied on compresses or used as a bath, and has given very good results, especially in children. By this method at least ten times the amount of the drug must be used, and consequently the item of expense has to be considered.[1]

The following other salts of quinine are recommended, without, however, presenting any advantages over the sulphate.

The *bisulphate of quinine* is soluble in water, and is preferred to the simple sulphate by some (Bonaventura), on account of its affecting the stomach less. Symptoms of cinchonism, however, (roaring and ringing in the ears, deafness, beating, and throbbing in the head, etc.), supervene more quickly after its use, and it is therefore to be given in smaller doses. *Hydrochlorate of quinine* is more soluble than the sulphate, contains a larger number of effective ingredients (in the ratio of 83.6 to 74.3), and is therefore also more expensive. According to Binz, it is greatly to be preferred to the sulphate. *Tannate of quinine*, on account of its greater insolubility, has to be used in larger doses, as much as two to four times as large. It is distinguished for its less unpleasant taste and for producing less symptoms of intoxication, consequently it recommends itself for use with children and nervous women, as well as in the diarrhœal and diaphoretic forms of intermittent. *Valerianate of quinine*, a very soluble salt, is used in the intermittent neuralgias of nervous women. The *sulphotartrate of quinine* is said to be more effective than the sulphate, on account of its greater solubility (Mazza), and to cure obstinate fevers and tumors of the spleen, even when given in small doses (Barella).

---

[1] *Chevrey* (Gaz. des Hôpit., 1856, No. 70) cured two children, eight and ten years old, who were seized with intermittens perniciosa hydrophobica, by enveloping them in cloths saturated with a strong, warm decoction of cinchona, and changed every two hours. They were cured in seventy-two hours. He also cured a case of dysenteric intermittent, in an actress, by cinchona baths of eight hours' duration (10 lbs. 8 ounces to the bath).

The remaining salts of quinine that have been recommended, as the *urate* (Pereira, Armand), the *arseniate* (Taglioni), used to the one-fifth or the one-fourth of a grain daily, in divided doses, the *citrate* and *lactate*, the *chino-picric acid*, the *iodide of quinine*, and the *iodide of iron and quinine*, are not deserving of any further notice.

Among the remaining alkaloids of cinchona that are in use, *cinchonine* is worthy of mention, the sulphate of which is employed, and is rendered more soluble by the addition of a few drops of sulphuric acid. The dose is at least one-half larger than that of quinine, in spite of which its effect is still slower and more uncertain, and it is therefore only adapted to light cases. The intoxication observed by Moutard and Martin, as occurring within one-quarter to one-half hour after the administration of even small doses of cinchonine, and which consisted of severe frontal headache, a feeling of weakness, even to fainting, pain in the stomach, and vomiting, appears, according to Briquet,[1] to depend on the use of the powder, and its action on the mucous membrane of the stomach, inasmuch as these symptoms do not follow the use of the article in solution. Experiments have also been made with the *bisulphate of cinchonine* (Tourner), giving similar results. The *sulphate of quinidia*, in the same doses as that of quinine, has been given, with good results, by Wunderlich. Sufficient experience with regard to *cinchonidia* is still wanting. The effect of *quinoidine*, on account of the variable proportions of quinine, cinchonine, coloring matter, and resin that enter into its composition, is very uncertain, and it has to be given in two or three times as large doses as quinine. Chemically pure salts of quinoidine, according to Bernatzik, give better results, and are said to be but little inferior to those of quinine. The preparations used are, the tincture of quinoidine,[2] in drachm doses, solutions of the article with the addition of a little acid, the citrate of chinoidine, or the powder with one-fifth part of tartaric acid (Bernatzik).

---

[1] Bullet. de l'Acad. de Med., T. 25, Nos. 11 and 12.

[2] Quinodine, two parts; dissolve in alcohol seventeen parts and pure hydrochloric acid one part, and filter.—*German Ph.*

Cinchona bark in powder or in decoction, the tinctura cinchonæ, or the tinctura cinchonæ comp., as well as other fever tinctures containing but little quinine, stand far behind the latter in point of efficacy, and are only to be recommended as tonics after a cure, or for the purpose of preventing relapses.

Aside from the prophylactic action of quinine, its most favorable effects are to be seen in simple intermittent fevers, of the quotidian or tertian type, and in certain masked forms. Its efficacy is less uniform even in the quartan form, as well as in pernicious fevers with serious local manifestations, and it is least effective and reliable in the remittent and continued fevers. Nevertheless, it remains an established fact that, even in the last-named forms, quinine is the most potent weapon that we possess for combating fever.

Notwithstanding the well-known and well-established power of quinine in intermittent fever, cases do occur where no sufficient results follow its use. This sometimes depends on the small doses, given every hour or two hours, or at all events on its not being given in sufficient quantities, or it may be due to the use of inappropriate or bad preparations;[1] at other times it may be caused by a cachectic condition of the patient, with an enlarged liver and spleen, these conditions also running their course with paroxysms of fever. In such cases I have overcome the fever by the employment of iron and quinine and the application of the cold douche. These conditions are often associated with bad habits of life and insufficient nourishment, and cannot be overcome until the latter are removed. At other times hidden diseases with febrile movements—here in Holland generally incipient pulmonary tuberculosis — are misinterpreted by both patient and physician. Occasionally quinine fails to do its work, without our being able to account for this failure, and we are obliged to have recourse to other means at our command.

The numerous investigations that have been made in the course of time on the effects of quinine have resulted in establishing its value more and more for the treatment of certain symptoms that arise in febrile diseases. Notwithstanding all this, however, we have thus far obtained but little insight into the method of its producing these effects, whence it necessarily follows that all attempts to arrive at a reasonable theory with regard to the way in which it acts in malarial fever have proved fruitless.

---

[1] I have often noticed that hard, old pills failed to produce any effect.

We need here merely allude to those phenomena which do not concern this part of the question, viz. : the exciting influence on the nervous system produced during cinchonism, the diminution of fibrine in the blood observed after large doses (Legroux), and the reduction in the secretion of bile. The features of the greatest importance to us are the modifications of temperature and of the condition of the pulse produced by quinine. And, unfortunately, it is just here that our knowledge is fragmentary. This much, however, seems to be certain, that quinine does not increase the giving off of heat. How far it acts on the centre that presides over the regulation of heat, or whether it does so at all, is uncertain, for Binz and Bouvier have observed a diminution of temperature even after cutting off connection with this centre. Neither are we justified on this account in taking refuge in the theory of diminished production of heat and oxidation within the body (Lewizky and Binz). The reduction in the rapidity of the pulse and in the tension within the arterial system are to be explained, according to Eulenburg and Lewizky, by the action of the drug on the heart; but it is not known whether this action is exerted on the motor nerves or on the muscular tissue itself. The investigations of Binz and others on the destructive power of quinine upon the lower organisms, infusoria and fungous growths which are known as inducers of fermentation and putrescence, are worthy of note. Unfortunately, however, these facts are not yet directly applicable to the question of malarial fever; for, as was stated under the head of etiology, none of these lower organisms have, as yet, been demonstrated with certainty to constitute the malarial poison. The fact, which is claimed by Piorry, Paget, and Kuchenmeister, and is doubted by Griesinger, that quinine reduces the size of the spleen in well persons, seems to be thoroughly established by recent investigation. Thus, Mosler has been able to demonstrate it by direct measurement of the spleens of dogs,[1] before and after giving hypodermic injections of quinine, and also by the reduction produced by means of quinine in an enlarged spleen, which had been rendered so by previous section of its nerve. It is thus far not known how this reduction is accomplished, whether quinine acts on the unstriped muscular fibres (Kuchenmeister), or on the nerves of the spleen, or whether it operates on the medulla oblongata, the irritation of which, as has been shown by Tarchanoff,[1] will produce marked contraction of the spleen, if the splanchnic nerves are intact. According to Binz, we may imagine the diminution in the size of the spleen to be dependent on a limitation in the development of colorless blood-corpuscles, inasmuch as he has observed that quinine hinders the amœboid movements of the same, and their escape through the walls of the blood-vessels in inflammatory processes. This also receives support from the investigations of Martin on inflammation of the liver, and the observations of Skoda to the effect that the infiltration, in intermittent pneumonia, is held in check by quinine. I should not like to deny that the action of quinine in malarial fever may be attributed *in part* to its influence in reducing the size of the spleen, as other remedies, which act in the same way on the spleen, such as eucalyptus globulus, gentianin, strychnine, the use of cold water, etc., have a reputation as febrifuges. That it acts in other ways besides this, however, is evident from the fact that the fever may disappear before the spleen is reduced, and that certain forms of fever, especially those known as masked, which run their course without any enlargement of the spleen, are cured by quinine. If we accept the theory of a specific poison causing malarial disease, we can, with our present knowledge, only say this much; that that poison can be rendered inoperative in the body by quinine, that is, it can be destroyed; for the manifold manifestations appearing in different organs during the various forms of malarial disease are so acted upon by this agent that, in the words of Griesinger, they disappear "not one after another, nor one as the result of the other, but simultaneously, as though modified and destroyed by some common central power."

---

[1] **Pathologie u. Therapie der Leukämie. Berlin, 1872.**

---

[1] Pflüger's Archiv f. Physiologie, Bd. VIII, Heft I, p. 97.

Ranking perhaps next to quinine is an article that has of late years been introduced into therapeutics, especially as a fever remedy, to wit, the leaves of the *eucalyptus globulus*, a large tree of the order *myrtaceæ*, which constitutes a considerable portion of the woods of Australia. Its medicinal use was first recommended in Spain, and it seems, both in its physiological and in its therapeutic action, very closely to resemble quinine; so that, from the experiments thus far made, which, it is true, are not very numerous, eucalyptus would appear to furnish us with a most admirable and quite inexpensive substitute for quinine. The trials made on inferior animals, and in which the ethereal oil extracted from the leaves, the eucalyptol, was used, show that the temperature of the body is lowered thereby, and that putrescent changes and alcoholic fermentation are prevented (Siegen,[1] Mees). According to Mees, the amœboid movements of the colorless corpuscles are arrested on the application of one-fifteenth to one-tenth per cent. of the remedy[2] (an effect which is produced by one-twentieth per cent. of quinine); and in inflammation they are prevented from escaping through the walls of the vessels, just as under the use of quinine, especially if the frog's mesentery be exposed directly to the fumes of the eucalyptol. Mosler has further demonstrated that after injection of the tincture, or its introduction by the mouth, the spleens of dogs, which had previously been exposed and measured, grew harder, more consistent, and smaller, and assumed a steel-gray color on their surface.

According to the observations of Groos, in Hungary, in the regions of the Theiss and Danube rivers, as published by Lorinser, he completely cured 43 out of 51 patients (84 per cent.) with this remedy, 11 of whom had previously been treated in vain with quinine; 5 out of 10 relapses were also overcome by the same means. Keller reports 432 cases, 310 of which ($71\frac{7}{10}$ per cent.) were completely cured, and 108 of which were followed by relapses; of 118 cases that were said to have been previously treated with quinine, 81 were completely restored under the use of eucalyptus. Canstan saw 15 out of 27 cases get well (55 per cent.); Bertherend had 11 recoveries in 12 patients (91.6 per cent.). Among 35 cases described by Mees, 13 (37 per cent.) were completely cured; among these were also quite a number who had previously been treated with quinine without effect; in 10 of these no change whatever was produced by treatment; 12 of them failed to continue the treatment, and the result was not known. The reports of Struve are equally favorable. Not so, however, are those of Papillon, who gave the bark of the tree as well as the leaves, in substance, made up with honey, to the amount of 180 grains a day, and who also used the alcoholic extract to the extent of 135 to 150 grains a day, with very little effect. Out of 17 cases, 6 got well without any treatment whatever, 2 under the use of eucalyptus, and in 9 quinine had to be resorted to. Similar results were obtained by Burdel,[1] in Sologne, among 123 patients with fevers of the quotidian, tertian, and quartan types: only 11 were cured without relapses, 22 for five days, 33 for nine days, and in 57 the treatment was without avail. The observations made in the clinic at Basle, and published by Fichter,[2] were likewise negative, inasmuch as the remedy, used in the form of tincture, was either entirely without effect or very uncertain in its action; whereas, in those same cases the fever was promptly subdued by quinine.

---

[1] Inaugural Dissertation. Bonn, 1873.

[2] *Binz* (Sitzungsber. der niederrh. Gesellschaft, Bonn, März 17, 1873) could not demonstrate this effect upon the blood-corpuscles.

It appears, then, from what has already been said, that eucalyptus globulus possesses virtues as a remedy in fevers, and, indeed, in all types of simple intermittent fever, being most effective, according to Keller, in the tertian type (75.57 per cent.), less so in the quartan, and still less in the quotidian; serving a good purpose in obstinate relapses, especially when quinine has failed, and in acute and chronic tumors of the spleen. It is distinguished by the rapidity of its action (usually preventing the third attack), has no disagreeable after-effects, tastes better than quinine, and is far cheaper.

The great lack of uniformity in the results arrived at under this treatment doubtless depends, to a certain degree, on the employment of too small doses. The quality of the preparations used has also a good deal to do with it, as the trees that grow in their native soil yield more valuable leaves than those cultivated in Europe; the fresh, young leaves, too, are more effective than the old, dry ones. It also seems to me to make a difference whether the cases treated are new or old. It would appear, according to my rather limited experience, that the remedy is more applicable in old, protracted cases than in recent ones, therein resembling arsenic. This would be confirmed by the negative results in the recent cases of Fichter's, and the positive results in the older Theiss-fever patients of Groos; those reported by Mees were also almost exclusively cases that had lasted for months and years.

---

[1] Bullet. gén. de thérap., 1872, p. 402. Ref. in Medic. Centralblatt, 1873, p. 688.

[2] Deutsches Archiv f. klin. Med., Bd. XII, p. 508. I, too, as the result of my own experience, cannot at present join in singing the praises of this remedy.

Probably the most desirable of the *preparations* of eucalyptus is the alcoholic extract, made from the fresh leaves, simply cut up fine, as in this way none of the ethereal oil is lost. Lorinser prepares the tincture by digesting one part of fresh leaves with three parts of alcohol for fourteen days,[1] and gives two to three teaspoonfuls in light cases, and in severer ones larger doses. The leaves have also been used in the form of an electuary, or as an infusion, in doses of half an ounce to an ounce a day.

An important place in the list of remedies for malarial fever has long been accorded to *arsenic.* At the same time its efficacy is undoubtedly less than that of quinine, especially in fresh attacks of fever, where its effects are either uncertain or entirely negative.[2] On the other hand, it is of value in cases where quinine fails, in old, inveterate, and frequently relapsing intermittents and in malarial cachexia, as well as in neuralgias, which often yield to arsenic alone when all other remedies have proved unavailing. Of course, the individual circumstances of the patient must be especially regarded when prescribing this drug. It is contraindicated during the existence of disturbances of digestion, in extreme old age, or in pernicious fevers; long-continued use of the article is also to be avoided. A cure is sometimes accomplished in a few days; oftener, however, after it has been used for two or three weeks. During its use a supporting diet should be taken, and in case of cachexia, iron should be combined with it. The most desirable preparation is *Fowler's solution*, of which eight to thirteen drops should be given two or three times a day (one-sixteenth to one-eleventh of a grain at a dose, one-ninth to one-third of a grain during the day); the *acidum arseniosum*[1] is not as good a form to use.

---

[1] The tincture which was used by Mees in Groningen and Leiden is prepared by digesting one part of the dry, young leaves with eight parts of alcohol at 25 per cent.

[2] This inefficacy is ascribed by *Boudin*, the great advocate of arsenic, to the fact that physicians do not sufficiently regard the circumstances that modify the effect of the drug; thus, for instance, the doses should be larger in summer than in winter, etc. The most recent periods have again developed warm advocates of this remedy; *Sistach*, (Gaz. méd. de Paris, 1861), *Turner* (Med. Times, 1861, Sept. 28), *Legrand* and *Pichaud* (Virchow and Hirsch's Jahresber., 1866, II, p. 177). Some of them recommend *Boudin's* formula: Boil fifteen grains of arsenious acid in thirty-four ounces of water until it is all dissolved; then add, besides the water that has been lost, thirty-four fluid ounces of white wine, and give a tablespoonful four times a day (viz., one-ninth of a grain of arsenic at a dose).

The recommendations made of late years by Barrant, Lorenzutti, Tessier, Calvert, Declat, and Treulich in favor of the use of *carbolic acid*,[2] internally, in doses of from ⅙ to ¾ of a grain each, and amounting to 2¼, 4½, or 9 grains a day, during the intermission; or subcutaneously (according to Tessier, 1⅘ grains to a fluid drachm of water, of which 20 to 40 drops are to be used shortly before the attack), have not been sufficiently confirmed by more recent observers, such as Paluel de Marmon, Caisne, Marken, Curschmann, and Eisenlohr. This article, which was lauded by the first-named observers as most admirable in its effects, has been found by the last two named to be utterly inoperative, as it neither controlled the duration or the intensity of the fever, nor produced the slightest effect on the swelling of the spleen, although given in large doses. Is it not possible that here, too, recent cases respond to the remedy differently from old and inveterate ones? The good results from the use of *common salt*, or *sea-salt*, which are claimed by Piorry, Thomas, Maroschkin, and Pioch as following doses of ʒ ij to ℥ ss, or even ʒ vij twice a day, especially in lymphatic persons, have not been confirmed by Grisolle, Mucarin, and others. A considerable number of observations have been made, of late years, on the value of the *sulphites of soda* and *magnesia*, which were first introduced into the treatment of malarial fever by Polli. These articles are particularly reported on by American and Italian physicians (Mazollini,[1] Ranzoni, Turner, Baxter, Leavit, Hampton), some of whom estimate their value as being equal to that of quinine. Griesinger and others have not been able to obtain any results from their use. Every one admits that their action is slower, and therefore they are not to be used instead of quinine in pernicious fevers. The sulphite of magnesia is given in doses to amount to ʒ iij, or ʒ iij, ℈ij to ʒ v, in solution, during the day: the soda salt to amount to ℥ ss. to ℥ j a day.

---

[1] *Clemens* (Gaz. des hôpit., 1860, No. 11) recommends a preparation, which, it is claimed, can be used for years without detriment, viz.: ℞. Acidi arseniosi, potassæ carbonat., āā gr. lx; solve in aquæ dest. f. ℥ ix. Adde brominii puri gr. cxx. At first it must be frequently shaken, and is fit for use after four weeks, though it must always be kept from the light. Three or four drops are to be taken, in water, once or twice a day.

[2] *White* (Med. Times and Gazette, 1868, Oct. 3d, p. 105) recently reports that the laborers in the petroleum works at Makum, in Upper Assam (India) enjoy marked immunity from the malarial fever, which is very prevalent there; this has been thought to be due to the neutralizing influence of the carbolic acid contained in the gaseous petroleum exhalations on the malarial poison.

The treatment instituted by Currie, in 1805, of subjecting the patients to affusions of *cold water* during the attack, was afterwards further amplified by Priessnitz and Fleury. The former of the two had his patients rubbed off with cold, wet cloths during the attack, and during the intermission had them packed, rubbed, and given the half-bath. Fleury only applied cold douches of 54° to 57° Fahr. one or two hours before the attack, using a general shower-bath and a local douche directed against the region of the spleen, throwing a shower-jet of one inch and a quarter in diameter. By means of experiments on animals, using the cold douche or ice-compresses over the region of the spleen, the spleen being exposed to direct observation and measurement, as well as by the use of both methods in acute and chronic intermittent fevers, Mosler has confirmed Fleury's assertion with regard to the effect of cold water, especially in the form of a douche, in reducing the size of the spleen. But he can find no support for the further statements of Fleury, viz., that quinine can be entirely replaced by the use of cold water, and that the latter is even more effective than the former in old, obstinate cases with anæmia and with tumors of the liver and spleen, especially as it guards against relapses. According to Mosler, the power of cold douches in reducing the size of the spleen, as well as their influence on the intermittent processes and in preventing relapses, is inferior to that of quinine, and they can therefore not be considered as a substitute for this drug, but merely as admirable adjuvants thereto. Cold baths and affusions, for the purpose of shortening the attack, need not be used in simple intermittent fever, as they are disagreeable to the patient and of doubtful utility. In the pernicious forms, when danger threatens, they are sometimes of good effect, in addition to quinine treatment. For several years I have used the local cold douche in enlargements of the liver and spleen after intermittent, and it has done admirable service, in combination with quinine treatment, even in old cases. I use baths of 72° to 75° Fahr., seldom warmer, and of fifteen or twenty minutes' duration, with a local douche of from one minute to a minute and a half.

---

[1] In Canstatt's Jahresbericht, 1863, IV, p. 82, and 1864, IV, p. 67, this writer compares the effects of the sulphites and of quinine. Under the former there were 83 per cent. of recoveries, under the latter 59. Relapses took place, under the former, in 5 per cent. of the cases, under the latter in 44.5 per cent. Sequelæ, under sulphites 3.2 per cent., under quinine 19 per cent. Gastric irritability does not forbid their use.

The *alcoholics*, such as rum, brandy, etc., are of doubtfu value as direct febrifuge agents, and it is only in the lighte forms of fever that they seem to do any good. *Chloroform* ha been recommended by various writers (Serrano, Delioux, Alienza Clellan, and others), to be used during the chill, in doses of on drachm, either given but once or repeated several times at inter vals of one quarter to one-half hour apart. This is administere less with a view to breaking up the fever, than for the purpos of shortening the attack, as refreshing sleep is very likely to fol low the first or second dose. Where chloroform cannot be ad ministered internally, it may be used as an enema, with olive oi or glycerine.

The most effective of the remaining vegetable substances see to be, *piperine*, of which nine to fifteen grains are to be give during the intermission, and from which Griesinger has see good results in the recent attacks of young persons; and th *sulphate of Bebeeria*, in doses of four and a half to fifteen grains during the intermission. The roots and young shoots of the box-tree, long since known as a febrifuge, have also been used again of late. Mazzolini claims to have treated 308 cases, with 235 recoveries, by means of the *sulphate of buxine* (the alkaloid obtained from the leaves and seeds), in doses of from fifteen to twenty-two grains, in pills, during the intermission. Casati reports a rapid cure in thirty-seven cases out of forty-five. This article is decidedly cheaper than quinine, and is said to be without its disagreeable after-effects. The remaining vegetable substances that have been recommended are of inferior or doubtful efficacy, such as salicine, apiol (a liquid obtained by the digestion of parsley-seed with ether), cnicine, veratrine, strychnine, santonine, gentianine, capsicum seeds, etc. The external application of spirits of camphor or peppermint, oil of turpentine, and laudanum along the spine are of no use in combating the fever.

In the light grades of *remittent* and *continued* fever, with gastro-bilious disturbances, it may be well, in addition to a strict regimen, to give some of the vegetable tonics, as quassia or gentian, with the alkaline carbonates; and under some circumstances, emetics or purgatives, such as calomel, jalap, and rhubarb. But here, too, quinine, in daily allowances of fifteen, twenty-two, or thirty grains and over, is our main reliance, and must continue to be given until the end of the fever. This is still more the case in the severer forms, where the doses must be increased; the local morbid conditions that may here arise, such as bronchitis, diarrhœa, dysentery, etc., require special treatment. In great irritability of the stomach, quinine is best given in combination with morphine or opium, or in extreme cases it may be administered by enema or hypodermically. In case of severe vomiting we may, in addition to ice and effervescing mixtures, employ cold and counter-irritants over the region of the stomach. In cerebral symptoms, great restlessness and excitability, quinine and opium, combined with constant bathing of the head with cold water, or ice-compresses to the head, are advised. In adynamic conditions, blisters to the back of the neck, with other irritants to the skin, and internally, wine, brandy, ammoniacal preparations, warming of the bed, frictions, etc., must be employed. Brown recommends nitric acid, three drops every hour, largely diluted. In all remittent and continued fevers, even with severe cerebral manifestations, we must abstain from general or copious local blood-letting, as even without this the disease will soon assume an adynamic character. In the fièvre bilieuse hématurique, Veillard recommends, at the beginning of the disease, an emetic of ipecac, and purgative doses of calomel followed by neutral salts. Here, too, quinine constitutes the main item of treatment, and must be given in doses of thirty to forty-five grains, and in case of severe vomiting be introduced by the rectum; in addition, acids and tonics, and in case of severe pain or great excitability, opiates and antispasmodics. If it is possible, the patients, in all these forms of fever, should be removed from the malarious region, even during their illness.

In the *masked* fevers quinine is to be used in the intermissions, and where this fails, arsenic. In facial neuralgia I have often seen good results, especially in nervous women, from the use of the valerianate of quinine and from the citrate of caffeine. Sometimes these may be advantageously combined with opium or morphine, and the veratrine ointment applied externally. In anæmic persons we give iron; in robust subjects, with torpor of the bowels, a course at the mineral springs of Carlsbad or Marienbad is of admirable service. In intermittent cardialgia, where quinine alone fails and numerous relapses occur, I order it in combination with bismuth and nux vomica as follows: Sulphate of quinine, 75 grains; subnitrate of bismuth, 45 grains; extract of nux vomica, 2½ grains; made into 50 pills. Give 5 pills twice a day. In anæmic subjects, we give iron with quinine, and in very severe cases, apply a few leeches, and warm, narcotic compresses over the region of the stomach. In intermittent diarrhœa the tannate of quinine is used, with opium; in cases of colic, quinine with opium or morphine, and, if there is constipation, a cathartic of sulphate of magnesia or sulphate or phosphate of soda; in spasmodic cough or sneezing, valerianate of quinine, with large doses of valerian root, best given in the form of a tea, several cups of which, cooled, may be drunk in the evening; in epileptiform attacks, valerianate of quinine with bromide of potassium.

During convalescence, and for the purpose of avoiding relapses, it is of the greatest consequence that all excesses in eating and drinking, and all chances of getting wet or taking cold be avoided, and this is particularly true in convalescence from the severer forms of fever. The continued use of quinine, as well as of bitter and aromatic tonics, such as quinoidine, the tinctura cinchonæ composita, the extract of cinchona, is to be recommended. In severe forms, iron should also be given, and if associated with enfeebled digestion, bismuth, as well as small doses of rhubarb. The very best treatment, beyond all doubt, is removal to some elevated non-malarious region. Those who, after suffering from fever, return from a southern (tropical) climate to colder latitudes, should dress warm, wear flannel next to the skin, and keep their feet well clad, for the purpose of avoiding colds. It is also well that such a return should take place during the summer months, from July to September. After arriving at home, special attention will have to be given to the tumors of the liver and spleen that still remain.

In primary *chronic malarial infection*, it is of great importance to avoid all predisposing causes, especially getting wet, taking cold, and a prolonged stay in marshy and unwholesome places. It is also of the utmost consequence that one should occupy a good, dry house, and, above all, such a bedroom. In addition to this, quinine, quinoidine, and compound tincture of cinchona, or the wine of chinchona—and in anæmic cases iron—should be continued for some time. Frequent cool baths, with douches to the region of the spleen; a good, strong, somewhat highly seasoned diet; good Bordeaux wine, or, for those in more moderate circumstances, small quantities of brandy, aid the cure materially. If the symptoms are more marked, we should give, daily, one larger dose of quinine (nine to twelve grains), which may best be given at evening.

In the treatment of *enlarged spleen* and *liver*, quinine is to be given as long as the thermometer shows even slight febrile movement, and on account of the anæmia that is often present, it had better be combined with iron.[1] In addition to this, I have learned to regard cool baths, with the jet douche over the region of the spleen and liver, as the most effective treatment; river, sea, and surf bathing are also of benefit. After all febrile movement has subsided, alterative remedies are most applicable; iodide of potassium, or, better yet, the syrup of the iodide of iron, or Blancard's iodide of iron pills, which are very popular in France and Holland; muriate of ammonia, or muriate of iron and ammonia; when the bowels are confined, aloes, or rhubarb with the alterative vegetable extracts (ext. of chelidonium, of taraxacum, etc.), and various mineral waters. Sometimes the simple chloride of sodium waters are sufficient (Homburg or Kissingen); the alkaline saline springs, however, are better (Karlsbad, Marienbad); and in disturbances of digestion and anæmia, the saline iron springs (Elster, Franzensbad, Rippoldsau, etc.), combined with mud baths, where these are to be had. Where pecuniary considerations forbid a journey to the baths, I order the artificial Karlsbad salts (one to two teaspoonfuls in half a winebottleful of hot water, of the temperature of 122° to 126.5° Fahr.) to be taken in the morning before eating. I fully agree with Griesinger in the opinion that this treatment is of more avail in swelling of the spleen only, than in those cases where the liver is likewise tumefied, inasmuch as the latter implies a higher grade of disease and our means of combating it are less effective. Pain in the region of the liver or spleen may call for local blood-letting, anodyne poultices, or the Priessnitz compress.

---

[1] *Machiavelli* recommends the chloride of bebeerine for the treatment of malarial enlargement of the spleen, to be given to the amount of 3½ to 15 grains daily, in alco-

Acute *dropsy*, as well as *albuminuria* of recent date, may often be promptly suppressed by the use of quinine and iron, and a good nourishing diet, to which, in the former case, mild vegetable diuretics may be added.

Advanced cachexia demands, in addition to careful dietetic treatment, a removal of the patient from the malarious district to a high, warm, mountainous region, exercise in the open air, light, nutritive diet, and warm clothing. The medicinal treatment consists in the use of quinine and iron, or arsenic, and,

---

holic or watery solution, or hypodermically. He saw, without any disagreeable results from its long-continued use, 26 complete cures among 41 cases; several others were materially improved, and only one died.

according to Keller, of the tinctura eucalypti globuli, in small doses, morning and evening, in addition to cool baths, with the jet douche over the liver and spleen, and general shower-baths.

All further complications and sequelæ demand special treatment.

EPIDEMIC
CEREBRO-SPINAL MENINGITIS.

ZIEMSSEN.

PIDEMIC CEREBRO-SPINAL MENINGITIS.

eneral works.

irsch, Handbuch der historisch-geographischen Pathologie, Bd. II, p. 624 et seq.—*Wunderlich*, Handbuch der Pathologie und Therapie, II Aufl., Bd. III, Abth. 1, p. 505 et seq.—*Häser*, Geschichte der epidemischen Krankheiten, II Aufl., 1865, p. 684 et seq.—*Leyden*, Klinik der Rückenmarkskrankheiten, Bd. I, 1874, p. 410 et seq.

pecial works.

rance. 1805. *Vieusseux* (Geneva), Journ. génér. de méd., XXIV, p. 163 (Hufeland's Journal, XXI, p. 181).—1814. *Comte* (Grenoble), Journal génér. de méd. de Sédillot, 1816.—1822. *Pratbernon* (Vesoul), Journ. génér. de méd. de Corvisart, Tom. 82, p. 74.—1837-1851. *C. Broussais*, Histoire des méningites cérébro-spinales, qui ont régné épidémiquement dans différentes garnisons en France depuis 1837-42. Paris, 1843 (Schmidt's Jahrb., XLIV, p. 254).—*J. Ch. M. Boudin*, Histoire du typhus cérébro-spinal ou de la maladie improprement appellée Méningite cérébro-spinale épidémique. Paris, 1854. (Complete history of the disease from 1837 to 1851).—1837-42. *Faure-Villars*, Histoire de l'épidémie de méningite cérébro-spinale (à Versailles, 1839). Paris, 1844.—Gazette méd. de Paris, VI, 441, VII, 465.—*Chauffard*, Revue médic., 1842, p. 190.—*Gasté*, Résumé clinique sur les méningites cérébro-rhach., etc. Metz, 1841.—*Simonin*, Recherches topogr. et méd. sur Nancy, 1854, p. 206.—*Rollet*, De la méningite, etc. Nancy, 1842.—Mémoires de l'académie de méd., X, Paris, 1843, cf. Gaz. méd. de Paris, 1844, Nr. 38, and Canstatt's Jahresbericht, 1846, IV, p. 107.—*Lefèvre* (Rochefort), Annales maritimes, 1840, Avril.—*Barnouin* (Avignon), Mémoire sur la méningite cérébro-spinale, etc. Avignon, 1842.—*Tourdes* (Strasbourg), Histoire de l'épidémie de méningite, etc., à Strasbourg en 1840 et 1841. Strasbourg et Paris, 1843. (Schmidt's Jahrb., Supplem., IV, 101.)—*Franke*, Die Epidemie zu Strasbourg. Wien, 1845.—*Wunschendorf*, Essai sur la méningite, etc., à Strasbourg en 1841. Strasbourg, 1841. (Schmidt's Jahrbücher, Suppl., I, 111, 117).—*Schilizzi* (Aigues-Mortes), Relation historique de la méningite cérébro-spinale, etc. Montpellier, 1842.—*Mahot* (Nantes), Epidémie de méningite, etc. Nantes, 1842.—*Gaultier de Clembay*, Mémoires de l'acad. de méd., XIV, p. 155.—1846-47. *Artigues* (Montpellier), Clinique de Montpellier, 1847, Nr. 194 and 195. Canstatt's Jahresb., 1847, IV, 74.—*Monchet* (Lyon), Gaz. méd de Paris, 1847, Nr. 15.—*Chapuy* (Lyon), De la méningite encephalo-rhachid. Thèse.

Paris, 1847.—*Corbin* (Orleans), Gaz. méd. de Paris, 1849. Nr. 24.—*Fall* (Nismes), Gaz. méd. de Montpellier, 1848, Mai.—*Maillot* (Lille), Gaz. méd. d Paris, C. III, 845.—*Levy* (Paris), Gaz. méd. de Paris, C. IV. 830.—*Gir* (Toulon), Lettre pathologique sur la méningite, pphl. Thèse de Montpellie Juin, 1851.—*Larivière* (Bordeaux), Journ. de méd. de Bordeaux, 1868, Aoû —*Bertrand* (Strasbourg), Relation de quelques cas de méningite, c. sp. Str bourg, 1868.

Italy. *S. de Renzi*, Sul tifo apoplettico-tetanico, pphl. Napoli, 1850.—*The* Rivista di varii lavori, pphl. Napoli, 1841.—*G. Spada* (Cervaro), Sul tifo ap plettico-tetanico, pphl. Napoli. 1840.—*A. J. du Maida*, Sul tifo apoplettic tetanico di Calabria. Filiatre Sebezio, 1842.—*G. Pagano*, Qualche paro intorno alla febbre soporosa convulsiva detta communamente Torticoll Napoli, 1842.

Africa. *Magail*, Rec. des mém. méd., LIX, 1845. (Schmidt's Jahrb., LIII, 31).—*Besseron* (Algier), Comptes rendus de l'acad. des sciences, 1847, Mai. (Ca statt's Jahresb., 1847, IV, 74).—*Sourier* et *Jacquot* (Tlemcen), Gaz. méd. d Paris, 1843, XIX, 4.—*Chayron*, Relation d'une épidémie de méningite céréb spinale en Afrique. Montpellier, 1850, 8.

Spain. *Thompson* (Gibraltar), Med. Times, 1845, April.—*Gillkrest*, Lon. Me Gazette, July, 1844, p. 455.

Denmark. Sundhetscollegiums Forhandlinger, 1846–49.

Great Britain. *Mayne* (Dublin), Dublin Quarterly Journal, III, 95. (Schmidt Jahrb., LV, 28, LVI, 46).—*Whittle* (Liverpool), London Med. Gazette, 18 IV, 807.—*McDowell* (Dublin), London Journ. of Med., 1851, Sept.—*Charl Murchison* (London), Lancet, I, 16, 1865, April 22.—*Wilks*, Brit. Med. Journ I, p. 427.—*Grimshaw*, Thermometric Observations on Epid. Cerebro-spin Mening., Med. Times and Gaz., 1868, Octbr. 24, p. 438.—*Johnson*, ibidem, 18 Octbr. 31, p. 469.—*Collins*, Report upon Epidemic Cerebro-spinal Meningit Dublin Quart. Journ., 1868, Aug., p. 170.

Sweden and Norway. Berättelse om Medicinalverket i Sverige or 1856–1860.—*Lindström*, Om meningitis cerebro-spinalis epid., etc. Lund., 1857.—*Wistra* Hygiea, XVIII and XIX.—Ofversigt of helso och sjukvården i Sverige, 185 60. Stockholm, 1863.—*Arentz*, Norsk Magazin for Laegevidenskaben, 18 XIV, 401.—Beretning om Sundhetsstilstanden in Norge i aaret, 1860. Chri tiania, 1863.

Netherlands. Nederlandsch Tijdschrift voor Geneeskunde, V, 15.

Russia, *Turkey*, *Greece*, and *the East.* *Küttner* (Petersburg), Petersb. med. Zeitschri 1869, XVI, p. 53.—*Hörschelmann* (Krimm), ibidem, XV, p. 265.—*Diaman pulos* (Smyrna), Wiener med. Presse, 1870, Nr. 34–36.—*Kotsonopulos* (Naupli 1869), Virchow's Archiv, Bd. 52, p. 65.—*Sandreczky* (Jerusalem), Berliner kli Wochenschrift, 1872, Nr. 20.

North America. *White*, New Orleans Med. Journ., 1847, Nr. 49.—*Chester*, ibid 1847, p. 314.—*Boiling*, ibid., 1847, p. 732.—*Ames*, ibid., 1848, p. 295.—*Dra* Treatise on the Principal Diseases of the Interior Valley of North Americ

Philadelphia, 1854, II, p. 751.—*Thomas*, Transactions of the Med. Society of the State of New York, 1858.—*Squire*, ibidem, 1858.—*Kendall*, ibidem, 1858 —*Saunders*, ibidem, 1859.—*Upham*, Hospital Notes in Illustration of the Congestive Fever so called, or Epidemic Cerebro-spinal Meningitis, etc. Boston, 1863.—*Watson*, Americ. Med. Times, 1864, May.—*Frothingham*, ibid., 1864, April.—*Woodward*, ibid., 1864, May.—*Lente*, ibid., 1864, July.—*Draper*, ibid., 1864, Aug., Sept.—*Black*, American Journ. of the Medical Sciences, 1865, Vol. 49.—*Burns*, ibidem.—*Lidell*, ibid.—*Stillé*, ibidem.—*Logan*, Philadelph. Med. and Surg. Reporter, 1870, May.—*Canada*, ibid., Febr.—*Horner* (Virginia), Philadelph. Med. and Surg. Reporter, 1871, July 8.—*Snively* (Maryland), ibidem, June 17.—*Williams*, ibidem, Dec. 9.—*Pitts* (Southern Mississippi), St. Louis Med. and Surg. Journ., 1871, May.—*Robinson* (Alabama), New York Med. Gaz., 1871, April 15.—*Howard* (Montreal in Canada), Med. Times and Gazette, 1872, Sept. 21.—For some observations on the sporadic occurrence of cerebro-spinal meningitis in 1872 in the United States, see Virchow-Hirsch, Jahresb., 1873, Bd. II, 1, p. 204 et seq.

ermany. 1823. *Sibergundi*, Beobachtung einer zuweilen mit Encephalitis complicirten Myelitis epidemica, welche in den Wintermonaten, 1822–23, in Dorsten a. d. Lippe geherrscht hat. Rheinische Jahrbücher für Medicin und Chirurgie, v. Harless Elberfeld, 1823, Bd. VII, p. 79.—1850. *Rinecker*, Verhandlungen der phys.-med. Gesellschaft zu Würzburg, 1850, I, p. 246.—1863 and 1864.—*Frentzel* (Berlin), Berl. klin. Wochenschrift, 1864, Nr. 21 and 22.—*Hanuscke* (Ottmachau in Silesia), ibid., 1864, Nr. 25.—*Hirsch* (and *Salomon*), ibid., 1864, Nr. 33 (Bromberg).—*Meyer*, Preuss. Medicinalzeitung, 1864, p. 348.—*Wunderlich*, Ueber Fälle von epid. Cerebrospinal-Meningitis in Leipzig. Archiv d. Heilkunde, Bd. V, p. 417, 1864.—*The same*, Weitere Mittheilungen, ibid., VI, p. 268, 1865.—*Ziemssen*, Meningitis cerebrospinalis epid. auch in Süddeutschland (autumn of 1864). Vorläufige Mittheilung. Centralbl. f. d. med. Wissensch., 1865, Nr. 2.—1865, and 1866.—*Bencke*, Epidemiologische Nachrichten, Archiv des Vereins f. wissensch. Heilk., 1865, Nr. 2 and 3.—*Ziemssen* and *Hess*, Klinische Beobachtungen über Meningitis cerebrospinalis epid. (Erlangen), Deutsches Archiv f. klin. med., I, pp. 72, and 346.—*Zülchauer* (Graudenz), M. c. epid., Berlin. klin. Wochenschrift, 1865, Nr. 18.—*Volz*, Aerztl. Mittheilungen aus Baden, XIX, 6, p. 45.—*Böhmer* (Pathol. Anat.), Bayr. ärztl. Intelligenzblatt, 1865, Nr. 39.—*Klebs* (Path. Anat.), Virchow's Archiv, XXXIV, p. 327.—*Salomon* (Holzminden), D. Klinik, 1865, Nr. 13.—*Schuchardt* (Hannover), In his Zeitschrift f. prakt. Heilk. u. Medicinalwesen, 1865, p. 263.—*Mende* (Eimbeck), ibid., p. 473.—*Rummel*, Mening. cerebrosp. epid. im Kreise Berent in Westpreussen. Neuruppin, 1865.—*Dotzauer*, M. c. ep. in Oberfranken, Bayr. ärtzl. Intelligenzblatt, 1865, Nr. 12.—*Merkel* and *Reuter* (Nürnberg), ibid., Nr. 13.—*Lindwurm* (Spessart), ibid., Nr. 21.—*Fronmüller* (Fürth), ibid., Nr. 26.—Amtlicher Bericht aus dem Regierungsbezirk Mittelfranken, ibid., Nr. 30.—*Seggel* (Forchheim), ibid., Nr. 46, 47.—*v. Rücker* (Hof), ibid., 1866, Nr. 16.—*Schweitzer* (Kronach), Die epidemische C.

M. Würzburg, 1866.—*Gowalowski* (Rastatt), Allg. militärärztliche Zeitung, 1865, Nr. 129.—*Niemeyer*, Die epidem. Cerebrospinal-Meningitis, nach Beobachtungen im Grossherzogthum Baden. Berlin, 1865.—*Pfeiffer*, Die Epidemie von M. c. ep. im Eisenacher Kreise, Jenaische Zeitschrift f. Med. u. Naturwissenschaft., II, p. 323.—*Gerhardt* (Thüringen), ibid., II, p. 338.—*Chvostek*, Wochenblatt der Zeitschrift der wiener Aerzte, 1865, Nr. 36.—*Rollet*, Wiener med. Wochenschrift, 1865, p. 751.—*Hirsch* (Danzig), Verhandlungen d. berliner med. Gesellschaft, I, p. 1.—*Lehmann* (Polzin in Pomerania), Deutsche Klinik, 1865, Nr. 47.—*Litten* (Neustettin), Journal f. Kinderkrankheiten, 1865, Bd. 44, p. 333.—*J. Burdon-Sanderson* (Danzig and vicinity), Brit. Med. Journ., 1865, May 6.—Lancet, II, 1, July, 1865.—*Merkel* (Nürnberg), Protrahirte Fälle, Hydrocephalus, Deutsch. Archiv f. klin. Medicin, I, p. 519.—*Meschede*, Deutsch. Klinik, 1865, Nr. 31.—*Virchow*, Discuss. in der med. Section der Naturforscherversammlung zu Hannover, Tagblatt 2 and 3.—*Orth* (Rheinpfalz), Würzburg, 1866.—*Mannkopf* (Berlin), Ueber Meningitis cerebro-spinalis epid. Braunschweig, 1866.—*Hirsch*, Die Meningitis cerebro-spinal. epid. vom historisch-geographischen u. pathol.-therap. Standpunkte bearb. Berlin, 1866.—*Rudnew* and *Burger* (Petersburg), Virchow's Archiv, Bd. XLI, p. 73.

1867-1874. *Siegfried* (Königsberg), De méningitide c. sp. Diss. inaug., 1867.—*Bitter* (Hannover), Allgem. wiener med. Zeitung, 1868.—*Baxa* (Pola), Wiener med. Presse, 1868, Nr. 11, 33.—*Bonsaing* (Pola), ibid., Nr. 19.—*Pinner* (Pola), Wiener med. Wochenschrift, Nr. 30, 33, 51, 52.—*De Moulon* (Trieste), Du typhus tetanique. Trieste, 1868.—*Immermann* and *Heller*, Pneumonie und Meningitis, D. Archiv f. kl. Med., 1869, V, p. 1 et seq.—*Schuchardt* (Thüringen), Zeitschrift f. Epidemiologie, 1870, Nr. 1 and 2.—*Groos* (Thüringen), ibid., Nr. 6.—*Stadthagen* (Berlin), Epidemie des Winters, 1870-1871, Diss. inaug. Berlin, 1871.—*Eulenberg* (Köln), Berlin. klin. Wochenschrift, 1871, Nr. 6, 7.—*Silomon* (Bonn), Winter, 1870–1871, Diss. inaug. Bonn, 1871.—*Zeroni*, Aerztliche Mittheilungen aus Baden, 1871, Nr. 17-21.—*Ames-Dros* (Canton Berne), Correspondenzblatt f. d. schweizer Aerzte, 1871, Nr., 9.—*Dörner* (Munich), Diss. inaug. München, 1871.—*Reich* (Weinheim in Baden), Bad. ärztliche Mittheilungen, 1872, 21.—*Kratschmer* (Wien), Wien. med. Wochenschrift, 1872, Nr. 26–28.—*Mosler*, Neuropathische Entstehung der einfachen Harnruhr (Hydrurie) durch Meningitis cerebrosp. epid., Virchow's Arch., 1873, Nr. 58, p. 44.—*A. Maurer*, Croupöse Pneumonie und Meningitis cerebrospinalis bei Kindern im ersten Lebensjahre, D. Archiv, f. klin. Med., Bd. XIV, p. 47, et seq., 1874.

Affections of the Organs of Hearing, *Erhard*. Berliner klin. Wochenschrift, 1865, Nr. 38.—*Heller*, Zur anatom. Begründung der Gohörstörungen bei M. c. Deutsch. Archiv f. klin. Med., 1867, IV, p. 482.—*Knapp* (New York), New York Med. Record, 1867, Aug. 15, p. 341.

Affections of the Eye. *v. Graefe*, Verhandlungen der berliner med. Gesellsch. I, p. 58.—*Jacobi* (Danzig), Graefe's Archiv f. Ophthalmologie, XI, p. 156, 1865.—*Schirmer* (Greifswald), Klin. Monatsblätter f. Augenheilkunde, v. Zehender, 1865, III, p. 275.—*Knapp*, Centralblatt f. d. med. Wissensch, 1865,

Nr. 33.—*Kreitmair* (Nürnberg), Bayr. ärztl. Intelligenzblatt, 1865, Nr. 21, and 22.—*H. Wilson*, Diseases of the Eye in c. m. Dubl. Quart. Journal, 1867, May.—*Socin*, Beitrag zur Lehre von den Sehstörungen bei Meningitis, Deutsch. Archiv f. klin. Med., VIII, p. 476, 1871.—*Turnbull*, Observations on the Nature and Treatment of Diseases of the Eye in Cerebro-spinal Meningitis, Philadelph. Med. and Surg. Reporter, 1868, March 7.—*Knapp* (New York), Blindness and Deafness in consequence of Epidemic Cerebro-spinal Meningitis, New York Med. Record, 1872, Aug. 15, p. 341.

INTRODUCTION.

We use the term *epidemic cerebro-spinal meningitis* to define an acute diffuse inflammation of the pia mater of the brain and spinal cord with deposit of a fibrino-purulent exudation; the infectious character of the disease being shown by its epidemic occurrence, the nature and mode of its propagation, and by its course and lesions.

It follows, therefore, that this affection is to be separated from the diseases of the spinal cord, and classed among the infectious diseases. It is naturally allied, as Hirsch has shown, to those infectious diseases which, like dysentery, are characterized by a marked localization in some tissue or organ of the body.

Its *synonymes* are very numerous: cerebro-spinal typhus, typhus cerebralis apoplecticus; fièvre cérébrale, phrénésie, cephalalgie épidémique, méningite cérébro-rhachidienne, méningite purulente épidémique (France); spotted fever, congestive fever, (North America); febbre soporoso-convulsiva, tifo apoplettico tetanico, torticollo (Italy); nacksjucka, dragsjucka (Sweden); Genickkrampf, Genickstarre, Hirnseuche.

This multiplicity of terms may be explained partly by the protean variety of forms which the disease assumes in different epidemics, and partly by the fact that careful attention has been directed to the lesions only within the last ten years.

HISTORY.

For an exact knowledge of this disease we are indebted to the last few decades, during which a severe and wide-spread epidemic has appeared, affording an opportunity for careful clinical, anatomical, and etiological study.

We have reliable accounts of the affection as far back as the beginning of the present century. It is very probable that it occurred during previous centuries, but the fact cannot be ascertained with certainty, as Hirsch has clearly shown in his Historico-geographic Pathology. We may dismiss, therefore, the opinion of many writers, the French in particular, that the disease prevailed in earlier, indeed even in ancient times, and will begin with the small circumscribed epidemics which occurred in Europe at the commencement of the present century.

The first epidemic prevailed in Geneva, from February to April, 1805.[1] Then followed epidemics in Grenoble (1814), Vesoul (1822), and one in Dorsten, on the Lippe (1833). In Grenoble it was almost exclusively confined to the garrison; in Vesoul, to the civil population. The disease which prevailed in Dorsten in the winter of 1822–23 was described by Sibergundi as a "myelitis, sometimes complicated with encephalitis;" but the symptoms described correspond to those of cerebro-spinal meningitis, as has recently been shown by Häser,[2] and other trustworthy writers.

After a long interval the disease again appeared in France, in 1837, and spread as an epidemic in Bayonne, Bordeaux, La Rochelle, and other places. In the following years it occurred in Versailles, then in north-western and north-eastern France, everywhere spreading with increasing violence. In many localities it remained confined in a remarkable manner for many years to the barracks, and was transferred to other places by the marches of the troops. Paris received only a transient visitation. In 1849 it disappeared temporarily from France. About the same time with the more formidable developments in France, the malady entered Italy, and prevailed there from 1839 to the spring of 1845; afterwards it appeared in Algiers, where it continued from 1839 to 1847, especially in the central and eastern districts.

In 1844 a transient epidemic occurred in Spain, at Gibraltar.

---

[1] Vieusseux, l. c.

[2] Geschichte der epidem. Krankheiten, 2 Auflage, p. 691.

In Denmark in 1845–48; in Great Britain in 1846, especially in Ireland—in England there were only traces of the disease (Liverpool). After an interval of several years the disease appeared in the south and south-west of Sweden in 1854.

Nowhere did the epidemic meningitis show such malignancy, regularity, and persistence in its progress as in Sweden. In the course of seven years eighteen out of the twenty-four Swedish stadtholderships were invaded by the disease, and 4,138 persons died. From this excessive mortality we may approximately infer the whole number of the sick, which could not be accurately ascertained by the board of health. The proportion of fatal cases in many neighborhoods was stated to have been as high as 66.6 per cent., but the grand total could not have been so large. The most northern point which the disease reached in Sweden was in Sundewall, situated near the sixty-third degree of north latitude.

In Norway there was an epidemic in 1859 and in 1860; in Holland a transient one in the winter of 1860–61.

In the United States of North America the disease made active progress from the year 1842, visited most of the States at long intervals, and prevailed with the greatest intensity during the civil war.

During the last few years it has appeared in Canada (1870), and in the United States has shown itself, either in sporadic cases or in small epidemics, up to the present time.

As previously mentioned, Germany saw the new guest first in Westphalia in the winter of 1822–23 (Sibergundi), afterwards in 1851 in Würzburg (v. Rinecker). Other small epidemics of malignant "inflammation of the brain," which were observed between 1830–50, in southern and central Germany, probably also belong to this disease.

An invasion of more severity and longer duration began in 1863. It appeared first in Silesia, Posen, and Pomerania, reached the Mark in 1864, and about the same time entered Hannover and Brunswick, also Saxony (Leipzig), and Thuringia (Eisenach). At no place, with perhaps the exception of Bromberg, did the disease prevail as a severe epidemic.

In southern Germany, however, where it was first observed by me in Erlangen, in the beginning of July, 1864, the epidemic became much more alarming. Its first appearance probably dates somewhat further back, since in the winter of 1862–3, as appears from the reports of Immermann and Heller, autopsies were made of five cases of suppurative cerebral meningitis at the Erlangen Poliklinik.

Almost simultaneously the disease appeared in Nuremberg, and in 1865 it spread extensively in central, upper, and lower Franconia.

Further south there were unimportant offshoots of the epidemic as far as Munich, Landshut, and Augsburg.

In 1864–65 Baden and Hesse were visited.

In Austria there were only small and isolated epidemics; in Vienna in 1865, in Pola in 1866, in Lissa and Trieste in 1867.

In Russia also the disease appeared in small epidemics, which attacked especially St. Petersburg in 1864–68. In the beginning of the year 1868 it was also observed in the Crimea.

We have reports of small and mild epidemics in Turkey, Greece, and Asia Minor during the years 1868–72; also in Nauplia (1868–69), Smyrna (1870), and Jerusalem (1872).

In Germany there have been traces of the disease here and there during the last few years; in 1871 in Berlin, Bonn, Nuremberg[1], Fürth, Munich, and Baden; in 1872 in Vienna; in 187 traces in central Franconia again, some of which I investigated also in upper Bavaria (Munich). The disease appears to hav become naturalized with us.

ETIOLOGY.

Although the past twenty years have furnished very abundant material for the study of epidemic meningitis, and althoug all the data of value in regard to the origin and spread of th disease have been investigated in the most diverse places, an generally with great care, we still find ourselves in entire ignorance of the original source of the affection. The expressio of Chauffard,[2] in his account of the epidemic at Avignon from the year 1840, "L'étiologie de cette affection est resté enveloppée d'ombres impénétrables," has even to-day much significance. It is true that in some points we have recently reached positive results, but in regard to the most important etiological questions, we are still far from certainty.

---

[1] In Nuremberg, according to *Maurer* (l. c.), a small epidemic prevailed from Marc to September (inclusive), with the numbers 1, 3, 13, 9, 3, 1, and 3 for the respecti months.

[2] Revue médic., 1842, Mai, cit. by *Hirsch*, Monographie, p. 127.

It will not readily be denied at the present time, by any one conversant with the subject, that *the meningitis is an infectious disease.* Although it is the inflammation and exudation which first strike the eye and tempt us to regard the whole process as a simple inflammation, yet a more careful examination shows us that this opinion cannot be maintained.

The infectious nature of the affection may be inferred from the following considerations:—Occurring only very rarely and sporadically, until within the last few decades, the disease has since spread as an epidemic over whole populations—wide districts—in fact over a large part of two continents, progressing either uniformly or by leaps; the symptoms and lesions are similar in the mildest, the moderate, and the severest cases; derangements such as eruptions, enlargement of the spleen, alterations in the blood, and muscular degeneration, which are found more or less well-marked in most infectious maladies, are of almost constant occurrence in the meningitis; finally, the course of the disease is rapid from its beginning to its fatal termination, which often occurs within a few hours in the height of malignant epidemics—a fact for which no satisfactory explanation is found in the lesions, nor any parallel except in what occurs in the most severe infectious diseases.

The question here naturally arises, Whether a comparison of all the facts positively shows *the identity or relationship of the meningitis with other infectious diseases?* It is maintained by English, American, Italian, and many French physicians, that the illness is a *typhous disease*, identical with or allied to exanthematous typhus, or the so-called cerebral typhus.[1] But the critical investigation of this question by German physicians, especially by Hirsch,[2] has shown the untenableness of this opinion. Hirsch has properly called attention to the fact that the connection which unites the "typhous" diseases in a common class is really a very loose one, and that there is still less reason for admitting epidemic meningitis into this group when we consider that this affection, independently of its still obscure pathogenesis, presents no symptomatological or anatomical facts which can entitle it to be included under that broad and elastic generic term "typhus."

---

[1] Hence the synonymes spotted fever, tifo apoplettico, etc.

[2] L. c., p. 145 et seq.

In epidemic meningitis the lymphatics, the spleen, and the bronchial mucous membrane are not affected in a marked or constant manner; nor in their heterogeneousness and capriciousness do the eruptions of the disease present a complete similarity to those of typhoid fever or exanthematous typhus. Careful clinical investigations during the last few decades have shown that the behavior of the fever and cerebral symptoms, and the absence of a typical course, etc., indicate a wide difference between the general character of the meningitis and that of the individual typhous diseases.

So also it can be readily shown that epidemic meningitis has no connection with *malarial diseases*, or, in other words, that there is no identity or relationship between the infecting principle of meningitis and malaria.

The frequently well-marked intermitting character of the fever, the fitful progress of the inflammation and exudation, give to many cases a superficial similarity to intermittent fever and intermitting inflammations, such as pneumonia intermittens. It is chiefly for this reason that some have admitted a relationship between the two infections, or at least a modification of the meningitic process by malaria.

That this opinion cannot be maintained, is readily seen upon careful examination. In the first place, the meningitis by no means selects malarious neighborhoods, but rather sandy, dry plateaus—the high plateaus of central Franconia, with their sandy soil, afford a suitable illustration; secondly, it does not prefer the seasons of the year and conditions of temperature which are favorable to the development of malarious epidemics, but rather the winter; thirdly, when occurring in malarious regions it shows neither a greater malignancy of the cases, nor more frequently an intermitting type than it does in other places which are free from malaria; nor, finally, is there any other similarity to intermittent fever. The spleen is generally only slightly enlarged, more frequently of a normal size, even in the so-called intermitting cases (v. Ziemssen and Hess); enlargement of the liver, melanæmia, and other sequelæ of malarial fever do not occur in this disease, and quinine is always useless in the intermitting cases. Finally, the preference of the disease for the age of childhood is opposed to the idea of malarial influence.

Quite recently (1867) the dependence of the meningitis upon malaria was disproved by Bonsaing (l. c.), who noticed that the Pola epidemic did not seek out the notoriously malarious parts of the city; that during the epidemic, and after its disappearance, malarial fevers were almost entirely absent, and that quinine was without effect in the intermitting form.

If now we consider the influence of the other important causes of infectious diseases, we find that *climate* and *conditions of soil* are wholly irrelevant; since, as Hirsch has shown, the disease arises and subsides in the same form, with nearly the same mortality and independence of fixed season or weather, whether in the eastern hemisphere from the northern coast of Africa to sixty degrees north latitude, or in the western hemisphere from the coast of the gulf to the New England States. The tropical regions proper have hitherto escaped the disease.

The *season of the year* and *the weather* seem nevertheless to have an important influence. The disease generally occurs in *winter* and *spring*, yet the effect of temperature, moisture, and direction of the wind has not been accurately ascertained in detail. The cold of winter appears to have less influence than great variations of temperature and the moisture of the air.

Hirsch gives a very instructive tabular statement of those French and Swedish epidemics in regard to which the time of their prevalence has been accurately given according to the respective months. (See following page.)

As regards *individual predisposition*, *age* and the *external conditions of life* are of much, *sex* of little importance.

With respect to *classifications of age*, *childhood* suffers severely both in susceptibility to the disease and in mortality. No age is wholly spared—we have examined after death persons as old as seventy to seventy-seven years—cases beyond the age of forty are very rare, somewhat more common between twenty and forty years, but in the first two decades of life excessively frequent. In many epidemics only children under fifteen years were attacked

Epidemics prevailed.	France.	Sweden.		France.	Sweden.
In December...	26 times	19 times	In Winter	97 times	119 times
" January.....	32 "	45 "			
" February....	39 "	55 "			
" March......	30 "	65 "	In Spring	69 "	192 "
" April......	23 "	68 "			
" May........	16 "	59 "			
" June........	16 "	37 "	In Summer	30 "	64 "
" July........	7 "	16 "			
" August.....	7 "	11 "			
" September ..	8 "	8 "	In Autumn	30 "	22 "
" October. ...	10 "	6 "			
" November...	12 "	8 "			

In Sweden, out of 1,267 fatal cases of meningitis occurring during 1855–60, where the age has been stated, 889 were under 15 years, 328 from 16–40 years, 50 above 40 years (Hirsch).

For the districts of Carthaus and Berent (circuit of Dantzig) Hirsch has prepared the following report of the mortality at different ages: Out of 779 fatal cases, 208 occurred in the first year of life, 337 from 1–5 years, 151 from 5–10 years, 41 from 10–15 years, 16 from 15–20 years, and 26 over 20 years. As Hirsch remarks: the relative susceptibility to the disease at different ages is not correctly shown by the tables of mortality, since the fatality is much greater during the first years of life than later, and therefore the numbers during the first ten years of life are much too high.

In the circuit of central Franconia, according to the official report (Aerztl. Intell.-Bl., 1865, No. 30), from June, 1864, to date of report in 1865, 456 persons were attacked, of whom

257 were from 0– 9 years of age
126 " " 10–19 " "
41 " " 20–29 " "
32 " over 30 " "

Of the 42 cases occurring under our own observation

14 were from 0– 9 years of age
13 " " 10–19 " "
9 " " 20–29 " "
6 " over 30 " "

The *external circumstances of life* which favor the outbreak of epidemic meningitis are those bad hygienic conditions which furnish the soil for the deposit and further development of the morbific germ, viz., poverty, insufficient nourishment, and damp, overcrowded, badly ventilated, unclean ground floors. The better class suffer little from the disease. The overcrowding of dwelling and sleeping rooms, and the consequent loading of the air with animal emanations, perhaps also the saturation of the soil with garbage and the products of its decomposition, appear to be as powerful agents in the germination of the contagium as they are in that of cholera.

In no other way can be explained the *frequency of local epidemics in workhouses* and other densely populated localities. The numerous epidemics during the years succeeding 1830 and 1840 in France, which were confined to the military in the barracks, entirely or almost entirely without extension to the civil population; the epidemics in the workhouses in Ireland during 1846; in the French brothels and prisons; the accumulation of the sick in certain streets, in rows of houses, and in detached dwellings, as is observed in every epidemic, compel us to look for the collateral causes of the development of the morbific germ in the same direction where they are found on an extensive scale in the case of cholera.

To the same hygienic errors may also be attributed the repeated severe epidemics among the negro slaves in North America. In these cases the fault lies rather with improper vital conditions than with a difference in race. The influence of the latter upon susceptibility to meningitis is still very doubtful, and cannot be traced in the course of the epidemics in the western hemisphere.

We must not conclude, however, that errors in hygiene are the only source of the disease and of its epidemic development. Such an idea is opposed by the permanence of these conditions without the continuance of the meningitis, and by the presence of the same conditions in other places without the development of the disease; finally also by the repeatedly observed transmission of the affection, a point to which we shall return hereafter.

The influence of *sex* is shown by the fact that almost always men are attacked in larger proportion than women.

Bodily conditions do not appear to play an important part. Those who are attacked are generally healthy and strong children or young persons. Nevertheless, chronic invalids and sufferers from acute affections, particularly of the air-passages, are frequently the subjects of the disease. The details upon this point will be given hereafter under the head of complications.

Whether the poisonous material is a *miasm* or a *contagium*, or whether the epidemic meningitis is to be regarded as a *miasmatic-contagious* disease, are questions at present far from settled.

The majority of writers very properly reject the idea of contagiousness in the strict sense of the word. If the disease generally spread by transmission from one person to another, there would be as little doubt of the fact as there is of the contagiousness of small-pox, measles, scarlet fever, exanthematous typhus, etc. On the contrary, it appears very probable, from the course of the epidemics and from some very trustworthy observations of the transmission of the disease, that we have to deal with a morbific germ, which primarily arises in the human body, and infects healthy neighbors only when it has undergone a certain, still unknown, modification by means of cultivation in suitable intermediate individuals [Zwischenträgern].

This, in the main, is the view expressed by French physicians, who have traced the transmission of the disease from one garrison to others. Hirsch has collected convincing facts[1] in regard to this point, and has added reliable observations from Berlin and western Prussia. I will briefly give the most important of them.

According to Frentzel, the first case of the disease among the troops in Berlin was in one of the reserve of the Alexander regiment, who a few days before had come from Liegnitz, where the meningitis was then prevailing as an epidemic. In the second company of this regiment, to which he belonged, five more cases afterwards occurred; in the two companies lying on either side there were three cases in one, two in the other; in the four companies beyond these only one, and that the mildest case, while there was no illness at all in the second battalion, which occupied the same barracks, and was separated from the first by only a small court; the two battalions, as is generally the case, associating but little with each other, but sharing completely the same labors and mode of life, and eventually the evils of overcrowding.

---

[1] L. c., p. 150.

The observation cited by Hirsch is still more striking.

On the 8th of February, in the township of Sczakau, K., aged 20 years, was taken ill, and was nursed by a young woman, W., who had hastened to him from the village of Sullenczyn. After the death of K. his nurse returned home, and there died, Feb. 26, of meningitis. This was the first fatal case of the disease in Sulleneczyn, with the exception of a previous one on January 15. To the burial of this maid at Sullenczyn came the family of the farm-steward K., from the township of Potgass, accompanied by a servant, D., and the four-year-old daughter O., of the teacher R., in Potgass. After their return from the funeral, a little child of K. sickened and died within twenty-four hours, then the servant D., who died March 4, and, finally, the girl R., on March 7.

Such observations, and others like them, made by the physicians of upper Franconia, in regard to the transmission of the disease, confirm the supposition of a poisonous material conveyable by intercourse, just as has been proved to be the case in cholera.

Whether it be, as in diphtheria and relapsing fever, a parasitic agent, which in some way gains access to the soft investing membranes of the central nervous system, and there excites exudative inflammation, as the diphtheritic germ probably does in the throat, is a question which must for the present remain unsettled. At all events, more searching study must be devoted to these points in future epidemics.

PATHOLOGY.

General Description of the Disease.

The meningitis almost always begins abruptly without a definite prodromal stage. In the epidemic observed by us we looked carefully for prodromata, but found them in only five out of forty-three cases. In these five cases there were headache, exhaustion, nausea, anorexia, and flying pains. Previous to the sudden outbreak of the disease there was generally an interval of several hours, during which the patient felt perfectly well.

The initial symptoms almost always present a furious character: a violent chill or chilliness, extreme malaise, which drives the patient at once to bed, raging headache, and free vomiting which recurs whenever he rises. In severe cases there occur a the start loss of consciousness, coma or delirium, convulsions, an the characteristic stiffness of the neck, which in a few hours ma develop into a tonic contraction of all the extensors of the spina column. In moderate cases there is only the stiffness of the neck the patient is not completely unconscious, but somnolent, tossin on the bed in continual restlessness; on being spoken to o questioned, he immediately awakens from his stupor and give answers which are generally correct but not very intelligible His most constant and loudest complaint is of the headache, an even when quite unconscious he shows the intensity of the pai by groans and by pressing his head with his hands. The face i more frequently pale than congested, and the expression of th countenance is that of profound suffering.

The disease develops rapidly to its full intensity. The feve is generally moderate, very irregular, and without typical cha acter. The pulse is either quite normal or moderately quickened but undergoes great and rapid changes in frequency. Durin the active delirium, which alternates with unconsciousness, an while the restlessness still continues, the skin becomes generall hyperæsthetic, and afterwards the other soft parts and the joints so that every movement of a limb, even the raising of a fol of the skin, causes loud expressions of pain. Then follow *cuta neous eruptions;* first herpes on the face, not infrequently o the extremities, sometimes symmetrically arranged on both side of the body; then erythema, roseola, urticaria, and petechiæ From the third to the fifth day the tongue becomes dry an cracked, the appetite fails, the bowels are constipated, mor rarely there is diarrhœa, and often the discharges are involun tary.

If the attack is to terminate fatally, the symptoms of irrita tion of the nervous system subside, and those of depressio increase; the patient lies in complete unconsciousness, there i little or no reaction, the urine and stools are passed involuntarily, the pulse becomes very rapid—towards the end too rapid to count—small, and scarcely perceptible, the temperature shows high elevations, there are convulsive muscular movements, paralysis of the cranial nerves, paresis of one-half of the body, general convulsions ending in profound coma, and finally death closes the scene.

If the disease run a favorable course, the symptoms of depression are not marked, or if they are, it is only for a short time. The patient complains continually of pains in the head, spinal column, and extremities, even on the slightest movement, and generally lies motionless in one position; the eyes are intolerant of light, the ears of noise; the vomiting subsides after five or six days; the headache and stiffness of the neck gradually diminish, and convalescence begins generally in from one to two weeks, sometimes later, but may be protracted for a long time by pains in the spinal column and extremities, and by disturbances in the organs of sight and hearing.

So different from this general outline of the severe and mild cases of the disease is that of the most severe and mildest types, as well as that of the intermitting form, that it is necessary to devote separate attention to these latter varieties.

1. *Meningitis cerebro-spinalis siderans* (Hirsch)—the méningite foudroyante of the French. The disease begins in the midst of perfect health, with a shaking chill, quickly followed by loss of consciousness, convulsions, and contraction of the neck. Death ensues in a few hours. According to the descriptions of the French writers, the patients in rare instances fall in the street, are struck down, as it were, in a moment, and are brought dying to the hospital.[1]

As an illustration of this form, take a case which occurred in the epidemic at Erlangen.

---

[1] "Tout à coup, au milieu de la santé la plus parfaite, des hommes pleins de jeunesse et de force étaient atteints des accidents les plus graves; ils succombaient en peu d'heures sans qu'aucun trouble fonctionel eût précédé cette subite invasion. Des militaires ont été frappés dans la rue, à l'exercise, dans les casernes, pendant leur repas, ils tombaient comme foudroyés, et l'on transportait à l'hôpital dans un état désespéré des hommes, qui peu auparavant faisaient leur service sans se plaindre."—*Tourdes*, l. c., cit. by Hirsch, p. 44.

Margaret Eckart, aged 8 years, was a feeble child from birth. Later in life sh was noticed to be deficient in intelligence and memory. Frequently without an external cause she fell into fits of weeping. For some years she had suffered fro attacks of severe headache. The mother of the child died of pulmonary tubercu losis.

On the afternoon of April 22 the child was suddenly taken ill with severe head ache while playing out of doors, and came home complaining and weeping. Aft being put to bed she suffered also from nausea, active vomiting, and vertigo. Th headache increased, the eyes became distorted, and the fingers of both hands firml clenched. This condition is said to have lasted about two hours, during which th patient remained apparently conscious, and often screamed loudly. *Evening*, s *o'clock.*—Present condition. Patient is gracefully formed, skin pale, muscles flabb and poorly developed. She lies quietly in bed, perfectly conscious, complaining headache and intense thirst; cheeks considerably reddened; neck not stiff. Ter perature 103.3° F. (rect.); pulse 100.

23, *Morning.*—Last evening several attacks of vomiting. Slept poorly, but th headache was slight, and is now entirely gone. The skin, especially that of th face, is very pale; moderately warm. Temperature 100.7° F. (rect.); pulse 100.

Towards *noon* patient left her bed feeling quite well; went out, and broug beer from a neighboring public-house.

About two o'clock, after having for some time amused herself with her sisters, sl suddenly became quiet, lay down upon the floor of the room, and complained severe headache. She was put to bed. After some time the "eyes became drawn and marked contractions of the hands and feet ensued, which soon passed into vi lent general convulsions, with continual groaning and screaming. Consciousness said to have been lost for only a short time.

About six o'clock in the *evening* the convulsions gradually ceased, and after as ing for a drink the child sank into stupor, and died half an hour afterwards.

Sectio cadaveris: A small amount of sero-purulent infiltration of the arachnoi and subarachnoid space in the brain and spinal cord, anæmia and œdema of th brain and spinal cord, bronchial catarrh and partial collapse of the lungs, swellin of the solitary follicles in the small and large intestines.

These cases occur in all epidemics, and most frequently at thei commencement. As a rule, they are fatal. Among forty-thre cases we have ourselves observed four, the duration of whic amounted to twelve, twenty-four, twenty-eight, and thirty hour All four terminated fatally.

2. *Meningitis cerebro-spinalis abortiva.* This abortive for of meningitis has for a long time been observed wherever ep demics have prevailed; a fact which furnishes additional pro of the infectious nature of the disease. Like other writers, w

have noticed, during the height of an epidemic, the lightest form in ambulant patients, who complained only of headache, stiffness and pain in the neck, and malaise, without being compelled to desist from their work. Such cases are naturally to be explained as rudimentary forms occurring on the outskirts of the epidemic.

As incidental complications of inflammatory affections of the lungs, pleura, and tonsils, we have observed symptoms which, though of a mild nature, were characteristic of meningitis, such as headache, with nausea and vomiting, sometimes preceded by chill, slight stiffness of the neck and of the whole spinal column, vertigo, unconsciousness, and slight elevations of temperature. Below are two such cases observed by us.

I. Johann Scholl, aged 19 years, journeyman cabinet-maker, was taken ill on May 12, 1865, at half-past six o'clock in the evening, in the midst of perfect health, with a shaking chill and severe headache. During the following sleepless night there were alternate chills, fever, and perspiration, and, in addition, stiffness of the neck. Towards morning the patient vomited a yellow, watery fluid, and had a consistent passage from the bowels. In the afternoon he was admitted into the University Hospital.

May 13, *Evening.*—Temperature 103.1° (rect.); pulse 80, full and hard; face slightly congested; pupils widely dilated, but reacting promptly; tongue moist, and somewhat coated. Forward movements of the head were difficult and painful. The dorsal portion of the spine was bent out of its natural curve, so as to be abnormally straight, and not easily flexible forwards. The lower vertebræ (from the seventh dorsal downwards) were of normal position and flexibility. No chest symptoms. Abdomen retracted, not painful. No eruption, no enlargement of spleen. Ordered twelve wet cups to the neck. Calomel gr. v, pulv. rad. jalap. gr. x.

May 14, *Morning.*—Slept quietly without dreams. Temperature 100.4° (rect.); pulse 80; less headache; neck still stiff and painful in forward movements; tongue much coated; some appetite.

Evening.—Temperature 100°; pulse 72; no headache. Stiffness of cervical vertebræ somewhat less. Vertigo. Two loose stools.

May 15, *Morning.*—Patient slept during the whole night. Temperature 99.2°; pulse 92, soft. About the chin and the corners of the mouth are some eczema vesicles. Patient can raise himself in bed, and remain in this position for some time without difficulty. Cervical vertebræ still somewhat stiff. Cervical glands symmetrically but slightly swollen. Vertigo. Constipation. Improved appetite.

Evening.—Temperature 99.9°; pulse 72. The head can now easily be bent forward, and the neck is no longer painful. Vertigo continues. General condition good. Constipation. Ordered ol. ricini.

May 16, *Morning.*—Temperature 97.2°; pulse 60. No vertigo. Subjectiv and objective condition normal. Discharged.

II. Jacob Nobenberger, eight years of age, son of a farmer in Hessdorf, a sle der boy and small for his age, was taken ill on the morning of May 25, with acu vomiting, headache, severe tearing pains in the neck, and immobility of the sam He was obliged to go to bed.

In the evening he was more cheerful, although the headache, according to l statement, is undiminished. On the following day (May 26) patient left his be Herpes on the lips, loss of appetite. Headache still present, especially towar evening. On the following days, during the period of headache, which recurr every evening, the hearing was so much affected that patient understood only wh loudly addressed.

On *May* 31, the seventh day of the disease, he presented himself at the clin after having travelled on foot an hour without much fatigue. The evening hea ache still continues. Pupils and pulse normal, herpes crusts on the lips. The tic ing of a watch was not heard on the left side at 2", on the right at 1". Nothi abnormal was found in the external auditory canal, nor in the membrana tympan

3. *Meningitis epidemica intermittens.* This form of meni gitis has also been observed in epidemics of the most diver character in France, Italy, Sweden, and Germany. It is disti guished by febrile attacks, recurring sometimes regularly in qu tidian or tertian form, with exacerbation of all the other sym toms, and by intervals of complete or almost complete freedo from fever and other indications of disease. The similarity severe cases of this kind to pernicious intermittent fever w noticed by Vieusseux. As has been already mentioned in tl chapter on Etiology, page 692, recent observations, especial those made in Germany, clearly show that this variety h nothing in common with malarial diseases, except the obje tive sign of an intermission of fever. I have myself, moreove demonstrated by systematic measurements of temperature, in tl Erlangen epidemic, the fact[1] that the alternation of intermissi and exacerbation is not always uniform, and that in this respe the resemblance to intermittent fever is more apparent than rea These intermissions appear either in the beginning of the di ease, both in the short and protracted cases, or in the stage deferverscence and convalescence.

---

[1] L. c., p. 401; see also the graphic representation of the course of the fever, page 714.

In the first case the short prodromal stage may consist of several attacks, as is shown by the case of Eckart, quoted at page 700; generally the intermitting character of the fever lasts for several weeks, and then changes to the continuous form, or terminates immediately in death or recovery.

In the second case the stage of defervescence is interrupted by more or less regular febrile exacerbations, which are often very severe, and are introduced by shaking chills, the thermometer rising to 104° Fahr. and upwards. The resemblance to the febrile attacks of pyæmia is very striking.

The significance of this behavior of the fever will be again discussed when I come to treat of the fever.

4. *Typhoid epidemic meningitis* is regarded by Hirsch, Tourdes, and others, as that modification of the disease in which, after the meningitis has lasted for some time, a so-called typhoid condition is developed, the patient lying soporose or with muttering delirium, with a dry, cracked tongue, sordes on the teeth, lips, and alæ of the nose, cold extremities, small, very rapid pulse, involuntary diarrhœal discharges, decubitus, and other similar symptoms.

Although it cannot be denied that there is a resemblance between the course of severe typhus and this variety of epidemic meningitis, yet to designate the latter as "typhoid" tends to identify it with the typhoid diseases, and to increase the confusion which it requires so much effort to remove. It is better to regard such cases as prolonged meningeal inflammations, in which the infection has been severe. Nowadays the typhoid forms of severe scarlet fever and measles are no longer regarded as scarlatinal or morbillous typhus, but rather as cases of violent infection.

Pathological Anatomy.

The lesions of epidemic meningitis are quite constant. The most important changes are found almost without exception at every autopsy, and vary only in the degree of their development.

The *emaciation* in the protracted cases is very great. The rigor mortis continues for a long time. Numerous hypostases of considerable size appear early, and sometimes elsewhere than on dependent parts.

The *skin* shows the remains of the different eruptions, especially herpes and petechiæ. The muscles, especially those extending along the spinal column, are reddish-brown or pale, and are found to have undergone granular degeneration (Zenker); the fat molecules being of such fineness that the fibres appear covered with a fine dust (Klebs).

In some cases multiple abscesses are found in both the subcutaneous and the intra-muscular *connective tissue* (Faure-Villars, Klebs).

The *calvarium* is frequently congested (in a punctate or linea manner), especially along the sutures.

The *dura mater* of the brain is often tense, smooth on it exterior surface, here and there united to the vitreous table sometimes specked with blood, the inner surface hyperæmic an adherent to the arachnoid.

In the *sinuses* thin fluid blood, loose or firm post-morte clots, rarely older decolorized thrombi are found.

The *arachnoid* is sometimes quite normal, sometimes hyperæmic or opaque, dry or viscous; after a protracted illness occasionally roughly thickened.

The *pia mater* is almost always hyperæmic, with capillar apoplexies, opaque and thickened by exudative infiltration. I can with difficulty be separated from the surface of the brain and often only by tearing the latter.

The more acute the course of the disease, the less free exudation is found between the pia mater and the arachnoid. In th fulminant cases the free exudation is entirely absent, and onl microscopic changes are found in the pia mater, in the for of a dense infiltration of cells, especially in the neighborhood o the vessels. Where free exudation is present in the subarachnoid space, it appears after an illness of from one to two day as a slightly cloudy mucous serum, or as a yellowish, whey-lik exudation.

If the disease have lasted from two to three days, the exudation is distinctly purulent, of a greasy, gelatinous, or firmer consistence, sometimes of a bloody tinge, and several lines in thick ness. It is deposited both on the convexity and at the base, especially along the course of the great vessels, in the folds and depressions of the surface of the brain, in the fissure of Sylvius, along the sulci, between the pons varolii and chiasma, and on the pons and cerebellum. In rare cases the whole surface of the brain is quite uniformly covered. The exudation consists of pus cells, free granules, fibrine, and mucine.

The *membranes* of the *spinal cord* are in much the same condition as those of the brain. The *dura mater* is often found separated from the vertebræ by extravasated blood, the *arachnoid* cloudy, and the *pia mater* thickened, and very adherent to the cord. The exudation appears here also at an early period as cloudy serum, but soon afterwards as a more or less thick fibrino-pus deposited in lines or as a layer, still later as a thick uniform stratum of pus, chiefly in the lumbar, less in the cervical region, and almost exclusively on the posterior surface of the cord. The roots of the spinal nerves are frequently bathed in pus. The anterior surface is much less frequently covered by the exudation; and when this is the case the whole cord is surrounded. The exudation is found posteriorly, principally because it flows to the dependent parts while it is still fluid (decubitus); and in the rare cases, where the cord is wholly imbedded—we have observed one exquisite case of this kind after the disease had lasted eleven days—the variation from the rule depends mainly upon the solidity of the exudation.

The *brain substance* appears sometimes congested, with punctiform hemorrhage and secondary development of small spots of softening; at other times, particularly when the disease has been hyperacute or very long-continued, it is juicy or œdematous, with a smooth, level surface, and of a watery appearance on section, whether in the cortex, medulla, or central ganglia. More rarely the substance is of a tough consistence.

In the majority of cases, especially those which are protracted, the ventricles contain considerable serum or a turbid, perhaps purely purulent exudation; the plexus and the ependyma are much injected, even ecchymosed, and covered with a layer of fibrino-pus. These conditions are found also in the third and fourth ventricles, though less frequently than in the lateral ventricles. The neighborhood of the ventricles is found to ha undergone hydrocephalic softening.

After a very long-continued illness the serous effusion m reach an enormous amount; resulting in eccentric atrophy the brain substance, flattening of the convolutions, and gr œdema of the brain and spinal cord. We also find shrinki and caseous degeneration of the exudation between the ara noid and pia mater, with opacity and callous thickening of th membranes.[1]

In the *substance* of the *spinal cord* the same changes found as in the brain (injection or anæmia, serous infiltration pulpy softening), but they are generally less marked, and l uniformly distributed through the organ.

As characteristic of the myelitic softening, Mannkopf found an abundant i tration of cells, which lay along the course of the vessels and extended with tl through the anterior and posterior fissures into the substance of the cord.

In a patient of Fronmüller, a servant girl aged fourteen yea who died after four days' illness, the autopsy made by revealed a large serous effusion, purulent exudation in the v tricles (including the fourth) and *dilatation of the central ca of the cord, which was filled with pure pus.*

Of the other organs, the *lungs* alone exhibit frequent chang such as simple hyperæmia and œdema, especially bronchioli in the lower lobes at their posterior surface, with a tough cat rhal secretion, secondary atelectases, and often lobular, m rarely lobar, pneumonic infiltrations. The pleuræ and the pe cardium are sometimes inflamed, covered with purulent exu tion, or ecchymosed. The *heart* is often flabby, and conta dark, thin fluid blood, with loose post-mortem coagula, less f quently firm fibrinous clots. Recent endocarditis is not of seen. The *liver* and *spleen* are generally congested, and of ve variable consistence. The liver cells often show an albuminous fatty cloudiness. The spleen is sometimes moderately enlarg The *stomach* frequently exhibits well-marked cadaverous soft ing, even in adults. In the *small intestine* the mucous me brane is normal or injected, even ecchymosed; the solitary follicles and Peyer's patches are swollen, and in rare instances ulcerated. In the *large intestine* we once saw marked lesions of dysentery. The *kidneys* are generally flabby and congested. The renal tubules are sometimes filled with fat granules and fibrinous casts. The *mucous membrane of the bladder* is here and there injected or ecchymosed. Purulent exudations are sometimes found in the joints.

---

[1] See *Ziemssen* and *Hess*, l.c., Cases XVI-XIX, and the section "Complications Sequelæ" in the present article.

The *auditory apparatus* is often affected, more rarely the *eye*, most rarely the organs of *smell* and *taste*. The disturbance in the functions of the latter senses is doubtless due to the pressure of the exudation upon the respective nerves at the base of the skull. The lesions in the eye are choroiditis, with consecutive detachment of the retina (Knapp); in the ear, if we may trust the few autopsies made hitherto, purulent inflammation of the labyrinth and tympanum (Heller, Lucae).

ANALYSIS OF INDIVIDUAL SYMPTOMS.

Headache may be regarded as an almost constant symptom, since it is rarely absent. It is generally very severe, extorting, even during coma, loud groans and sighs of pain. It causes also great restlessness; the patient frantically strikes out with his arms, and stamps with his legs, presses his head automatically, and moves it to and fro; finally, in some instances he becomes furiously delirious, springs out of bed, fights those about him, and requires forcible restraint.

The pain is situated sometimes in the forehead, sometimes at the back of the head or temples, occasionally it extends over the whole head, and is of a beating, boring, stabbing character; or the head feels as if compressed by a band. The seat of the headache does not always correspond to the situation of the inflammatory products found after death, nor is the violence of the pain in direct ratio to the intensity or danger of the disease. In many cases there are not merely marked remissions of the headache, but distinct intermissions, although generally the pain continues of uniform intensity, with slight daily variations. Its cessation during the attack is one of the most favorable symptoms, yet we must expect several exacerbations elevation of temperature, during convalescence.

According to our observations, the headache in ma stances continues for years after recovery from the meningi returns upon exertion of the body or mind, stooping, etc., accompanied by pain in the back and weakness of the limbs. These symptoms perhaps depend upon the roughening of the pia mater, which we have repeatedly found i sequent autopsies, and also upon the tendency to congesti inflammatory disturbances which is left behind in the pia from the previous inflammation and its results. We shall to these points when we come to consider the sequelæ.

Vertigo is frequently associated with the headache. I the convalescence the return of the vertigo and headache, had entirely disappeared, especially if associated with vo and convulsions, is a very bad sign, because it often indica development of hydrocephalus.

Vomiting is rarely absent. It is an initial sympto tinues generally not more than two days, and is excited b ments, especially by rising. In some cases it frequently during the whole course of the disease, is each time accom by increased headache, and prostrates the patient very because it interferes seriously with the assimilation of n ment. It appears to me probable that this return of th ache and vomiting is owing to a renewal of inflammation gestion.

It is scarcely necessary to mention that the vomiting regarded as only a symptomatic expression of the irrita the meninges and brain, and not as an indication of simp tric derangement.

Stiffness or *contraction of the neck* is the third c symptom, and gives such a characteristic expression to t ease that, as has been already mentioned, the affection is larly known[1] as "der Genickkrampf."

The *stiffness of the neck* is due to a more or less dev contraction of the deep cervical muscles—the trapezii are always unaffected. There are great variations in the de contraction—from a slight stiffness, felt only when the patient attempts to bend the head forward, to so great a retraction that the back of the head presents nearly a right angle with the spinal column. In the latter case the patient swallows with much difficulty, probably because the larynx is firmly pressed against the spinal column by the extreme tension of the anterior muscles of the neck (sterno-thyroid, thyro-hyoid and sterno-hyoid). This marked contraction generally occurs between the second and fifth days. The stiffness is rarely seen on the first day, and then only to a slight extent.

---

[1] In Germany.—TRANSLATOR.

In almost half the cases there is also more or less *contraction of the other erector muscles* of the spine—from a mere stiffness and straightening of some parts of the column, with disappearance of the spinous processes, and with the presence of pad-like projections of the hardened muscles on both sides, to complete *opisthotonos*. The latter condition, as far as we have observed, is rare, but complete *orthotonos* is frequent. The spine then becomes a straight shaft, and the convexity of the dorsal and concavity of the lumbar curves disappear. If we attempt to raise the patient, the body remains stiff, and the patient, in his violent efforts to bend himself, slips to the foot of the bed ; or we may succeed in getting him into a half-sitting position by means of some still remaining flexibility of the lumbar vertebræ, or by bending him at the hip-joints. The severe pains in the spine, induced by the strain, prevent him from long maintaining this posture.

Pleurothotonos, or *unilateral contraction of the spinal erector muscles*, has been observed (Levy, Hirsch), but is certainly very rare.

The contraction lasts a very variable time. In favorable cases it disappears in from two to three weeks, but not unfrequently continues much longer (four to six weeks) ; indeed we have several times seen convalescents going about with a rigid spine.

In rare instances the stiffness of the neck is entirely absent. I have myself seen some cases of this kind in which, at the autopsies, the anatomical changes, and particularly the extent of the inflammation in the spinal meninges, were not found to be at all different from those of other cases where the symptom was well marked. The absence of this symptom cannot be explained.

Pathogenetically, the stiffness of the neck is to be regarded a a *reflex contraction*, due to inflammation of the pia mater of th medulla oblongata, and probably also of the posterior column and posterior roots of the cervical portion of the cord.

Trismus and contraction or rigidity of other muscles, particularly those of the extremities, are rarely seen. Trismus i usually observed only in those who are dangerously ill an comatose, and is an unfavorable prognostic symptom.

Rachialgia, or *pain in the spine*, especially in the neck varies considerably in intensity and duration. Many patient with rigid spines are wholly free from this pain as long as the lie immovable on their sides, yet make loud complaints whe any attempt is made to bend the head or trunk.

In other cases the pain continues severe in whatever positio the patient lies.

Exacerbations and remissions of the rachialgia occur, a though less frequently perhaps than is the case with the cephalalgia, and sometimes the stiffness of the neck increases or remit synchronously with the pain.

Besides its situation in the neck, the pain affects most fr quently the loins, and more rarely extends along the whole spin

Pains in the extremities, especially the legs, are a freque symptom, and are caused or increased by concussion of t spinal column. They are described as darting with the rapidity lightning. Other parts of the body are also affected, the fund ment and the sterno-cleido-mastoid muscles, for instance, an sometimes the pains shoot quickly from one place to another.

Patients also frequently complain of a persistent pressure i the epigastrium, pain in the chest, and difficulty of breathing ; i one case, in fact, we observed repeated severe asthmatic attacks

A universal, or at least a widespread, *hyperæsthesia of t skin, the other soft parts, and the joints* is one of the more in portant symptoms of the spinal meningitis, but is by no mea a constant one. Hirsch, Fräntzel, and myself failed to find in many cases. When it is present, it occurs most frequentl in the lower extremities, occasionally at the same time in th trunk and upper extremities. It is so severe that every movement of the legs, every pressure on the surface of the body, even shaking the bed while the patient is profoundly comatose, excites loud expressions of pain, and even reflex convulsions. Associated with this condition is often found great sensibility of the senses to light, odors, etc. As Hirsch has correctly observed, this general hyperæsthesia interferes very much with the examination of the patient.

Partial anæsthesia of different parts of the skin has hitherto been noticed with much less frequency.

Cutaneous lesions are very common. Copious eruptions are a marked symptom in some epidemics, while in others they are less frequent. It would be difficult from our own observations to understand why the American physicians called the disease "spotted fever,"[1] were it not that roseola and petechiæ are more frequent symptoms of epidemic meningitis in America than in Germany.

Of all the different eruptions, *herpes facialis* is the most frequent. It begins generally in the neighborhood of the mouth, upon the upper or lower lip, sometimes on both sides, and extends to the cheeks, nose, ear, and eyelids. I have never seen herpes in other diseases spread so widely as in meningitis. It is by no means rare to see one or both sides of the face covered with vesicles or crusts. Irregular extensions of the eruption often take place very late in the disease, even in convalescence (in the sixth or eighth week, Hirsch). Tourdes and myself have observed that it generally begins from the third to the sixth day. With our present information no prognostic significance can be ascribed to the eruption.

Petechiæ are the next most frequent eruption; then follow *roseola*, *erythema*, *urticaria*, *erysipelas*, and *sudamina*. Sometimes a patient presents three or four varieties. Vieusseux, Faure-Villars, Upham, and others have called attention to the ominous significance of the ecchymoses appearing in the latter

---

[1] Exanthematous typhus was formerly known by its other name, spotted fever (fleckfieber), and hence this designation of the meningitis has led to an unfortunate misconception on the part of American and English physicians as to the nature of the new disease.

part of the disease, and the extensive hemorrhagic suffusions
the skin, which resemble the livid spots of the cadaver.

It is interesting to notice the frequent symmetry of the er
tion on both sides of the body. Not only on the face and tru
but also on the extremities, the herpes is often found develo
bilaterally in exactly corresponding situations. Thus, we h
seen herpes present at the same time on the joints of both han
urticaria on both legs, and petechiæ on both shoulders, etc.

The symmetrical appearance, variety, and frequency of
eruptions, as well as the hyperæsthesia, point very strongly t
trophic disturbance of innervation in the brain and spinal cord.
explanation similar to the one now accepted as probable, if
certain, in regard to the lesion present in non-meningitic zoste

Besides the symptoms above mentioned, *fever* is also a c
stant accompaniment of the disease.

The *behavior of the bodily temperature*, as shown especia
by Wunderlich and myself, is irregular.

Generally the temperature is quite high, not always at the
ginning, but first on the second or third day; sometimes, howe
it rises rapidly after the initial chill. In severe cases, especi
towards the end, it is sometimes as high as 107° F., and high

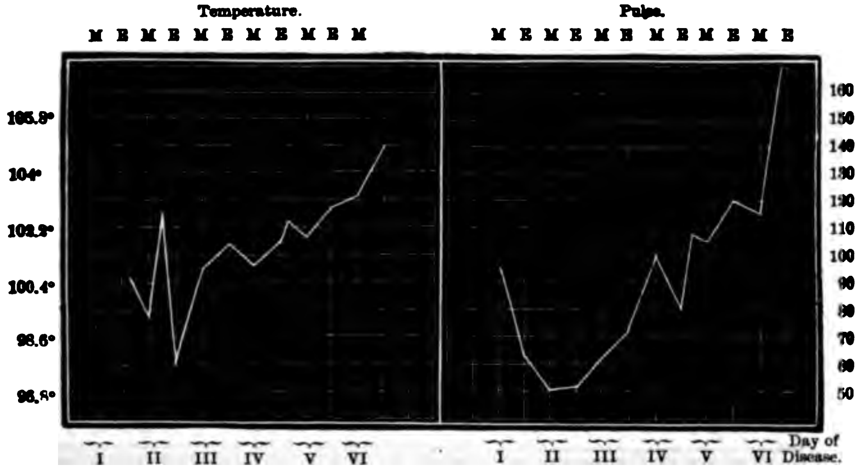
Generally the medium temperatures are from 100.4° to 1
with very irregular variations above and below these poi
often interrupted by long-continued normal temperatures, w
the other symptoms continue unabated. Moreover, if we c
pare the temperature tables of numerous cases, we find that o
a few of the curves resemble each other.

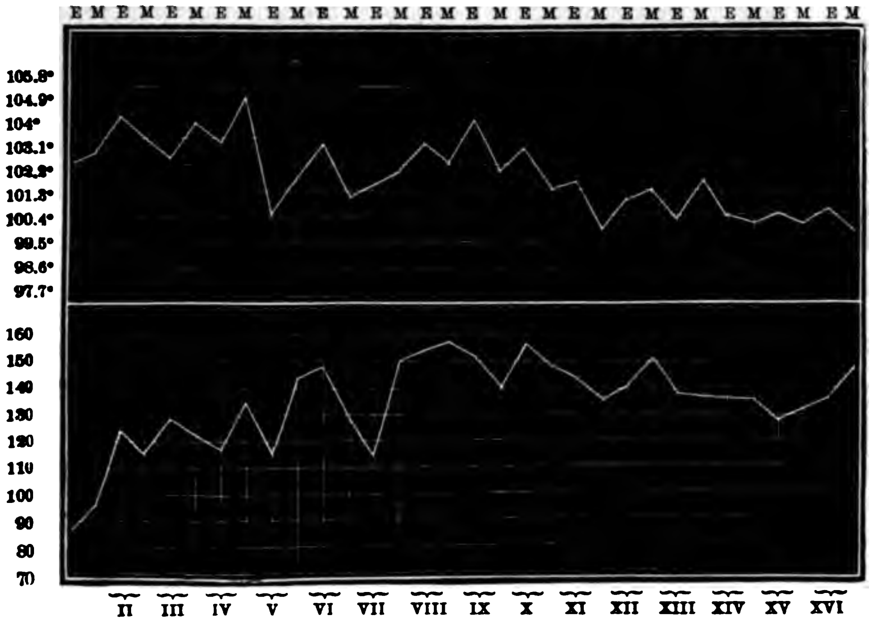
Below are found some of the curves, together with brief abstracts of the c
illustrating the course of the temperature and pulse in severe and mild cases.

I.—*Severe Form.*

L. W., aged 15 years, a plasterer's apprentice. Access abrupt, with c
cephalalgia, vomiting, trismus, tetanus of cervical and spinal muscles. Conju
vitis, hyperæsthesia of skin. After the fourth day, herpes facialis, roseola, erythe
urticaria, and petechiæ on the extremities. Moderate fever, with retardatio
pulse. Furious delirium followed by sopor. With rapid elevation of tempera
and pulse, death ensued on the seventh day of the disease.

Autopsy.—Purulent cerebro-spinal meningitis. Remains of old pleurisy perihepatitis. Partial atelectasis of lungs, and lobular pneumonia. Cadaverous softening of the stomach and diaphragm (five hours after death). Suppurative tendo-synovitis in the left hand. Areas of degeneration in the spinal and recti abdominis muscles. Ulceration of cornea.



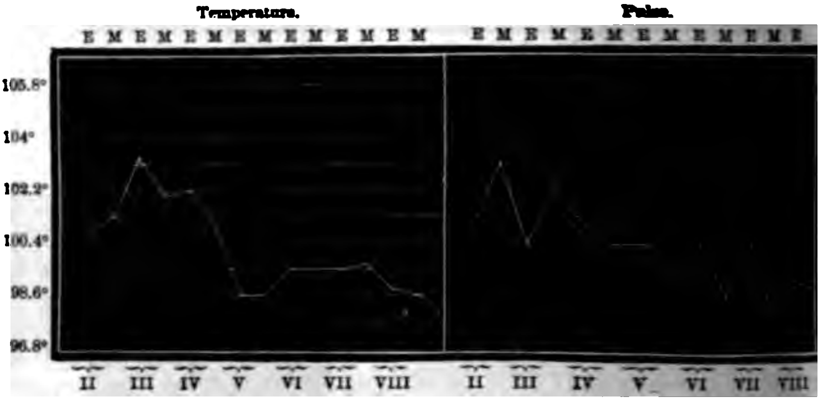


II.—*Moderately Severe Form.*

M. V., aged 12 years, daughter of a stocking-weaver. Access abrupt, with chill, and vomiting lasting during the first four days. Frontal headache. Stiffness a pain in the spine, jactitation, urgent thirst. Mind at first clear, afterwards delir and somnolence. Petechiæ on the second day. Herpes on the face on the seven on the thumb on the tenth day. Effusion into the right wrist-joint. Conjunc vitis and keratitis. Aphthæ. Temperature at first high, but gradually dimini ing, while the pulse becomes very rapid. Tedious convalescence. Duration of disease about six weeks.

III.—*Mild Form.*

C. St., aged 16 years, daughter of an umbrella-maker. After a prodromal st of two days' duration, patient was taken ill with pains in the head, extremities, epigastrium, nausea, vomiting, stiffness of neck, delirium, premature menstruati herpes on the fourth day, conjunctivitis, and transient fever. Improvement at end of the first week. Duration of the disease three weeks.

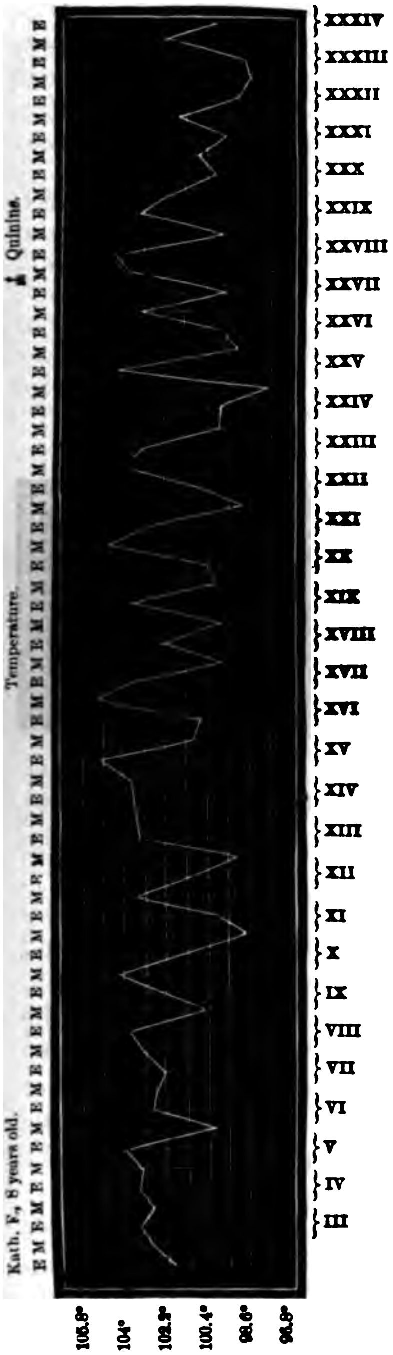


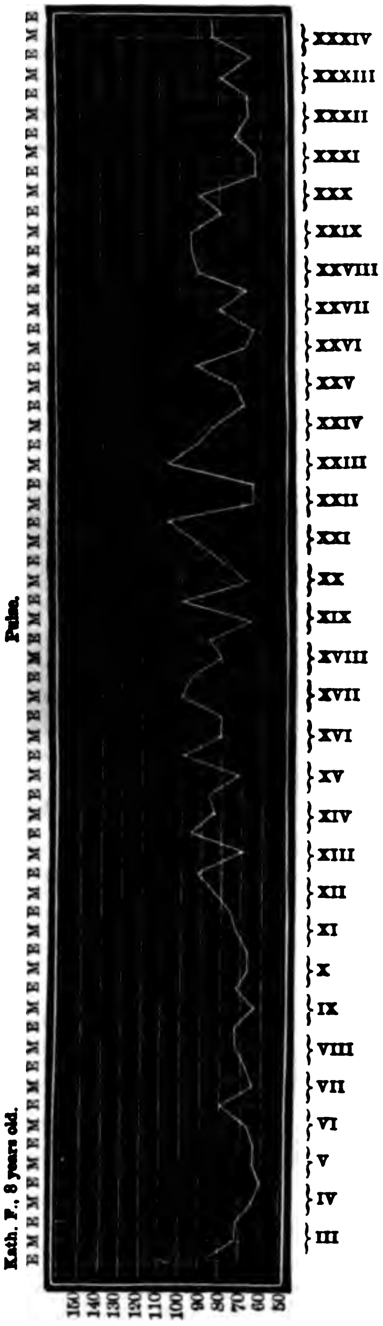
Intermittent Form.

In these cases the accurate use of the thermometer by means shows that regularity in the alternation of fever and a rexia which a superficial observation seems to indicate. T remissions and intermissions often extend over several days, that the graphic representation of the course of the temperat presents the appearance of great irregularity, sometimes even a mere recurrence.

Below are curves of intermittent cases:

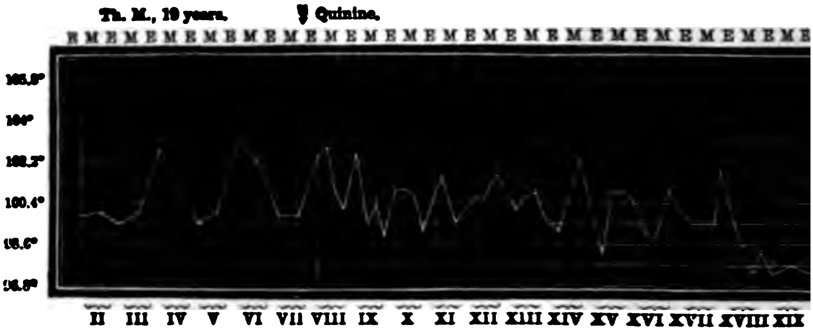
1. Catharine F., aged 8 years, daughter of a laborer. Access abrupt, with vo iting and cephalalgia. Chill, pains in the neck, difficulty in swallowing, stiff and tenderness in the neck and dorsal region of the spine. Sensorium clear, oc


Kath. F., 8 years old.
Temperature.
Quinine.
E M
III
IV
V
VI
VII
VIII
IX
X
XI
XII
XIII
XIV
XV
XVI
XVII
XVIII
XIX
XX
XXI
XXII
XXIII
XXIV
XXV
XXVI
XXVII
XXVIII
XXIX
XXX
XXXI
XXXII
XXXIII
XXXIV
105.8°
104°
102.2°
100.4°
98.6°
96.8°


Kath. F., 8 years old.
Pulse.
E M
III
IV
V
VI
VII
VIII
IX
X
XI
XII
XIII
XIV
XV
XVI
XVII
XVIII
XIX
XX
XXI
XXII
XXIII
XXIV
XXV
XXVI
XXVII
XXVIII
XXIX
XXX
XXXI
XXXII
XXXIII
XXXIV
150
140
130
120
110
100
90
80
70
60
50

sionally mild delirium. Epigastric pains. Herpes labialis on the fourth d Temporary polyuria and slight albuminuria. Pulse at first retarded, afterward variable frequency. Temperature very high at the beginning, later in the dis elevations apparently intermittent, preceded by chill, vomiting, and cephalal Cessation of these attacks in the fifth week. Recovery in the seventh w (Temp. curve, p. 715.)

2. Th. M., aged 19 years, student. Prodromata for eight days. Access ab with cephalalgia, vomiting, slight convulsions, unconsciousness. Neck some stiff. No fever during the first few days. Exacerbation on the fourth day, fever of short duration, followed by apyrexia and disappearance of the ma On the fifth, seventh, and eighth days the exacerbations recurred with marked s symptoms. Then followed daily exacerbations, but of less intensity and sh duration. No eruption. Complete cessation of febrile attacks after the eight day. Recovery. Duration of disease six weeks; of convalescence four weeks.



I have already shown that in these cases there is no t intermittence. In many of them the febrile attacks in the be ning and in the subsequent course of the disease are probal as it seems to me, due to the irregular progress of the inflam tion, and thus resemble what occurs in migratory erysipelas pneumonia ambulans.

In the retrogressive stage these attacks often present character of the absorption fever, so frequently seen during retrograde metamorphosis of purulent exudations on ot serous membranes (pleura, peritoneum). When the atta occur during convalescence they are more probably due to sli returns of the inflammation.

The *pulse* is generally as irregular as the temperature. frequency does not always correspond to the height of the te perature and the intensity of the other symptoms. Attention particularly called to the pulse curve on page 715, lest the di gence from the temperature curve there shown may be overlooked. When the inflammatory and febrile symptoms are of equal intensity, the pulse is sometimes normal, sometimes moderately, at other times considerably increased in frequency, but rarely retarded. Occasionally it varies thirty to forty beats or more within the course of a few hours; indeed, even within a few minutes, as Tourdes has observed, differences of as much as thirty beats may occur.

Abnormal slowness of the pulse is much less frequent than in tuberculous basilar meningitis. It rarely occurs except at the outset, and then in connection with lowered temperature, soon changes to increased frequency, and in fatal cases usually the pulse rises so high that it cannot be counted. Irregularity in rhythm is not uncommon. Continued rapidity is generally to be regarded as an unfavorable symptom.

In quality the pulse is either quite normal or of increased fulness and tension. When the symptoms of depression occur it becomes small and weak, and when at the same time extremely rapid, very difficult to count.

We now come to the consideration of certain disturbances which, though less frequent, are still not without significance.

Of those affecting the *central nervous system*, the following may be mentioned:—

General or *partial convulsions* are most often seen in the fulminant and comatose forms occurring in children, sometimes on one half of the body with paresis of the opposite side, sometimes alternating with tonic contractions. If long continued, they are generally an unfavorable symptom. Taking place in the retrogressive stage, they indicate the commencement of hydrocephalus (see Complications and Sequelæ).

Clonic convulsions also occur in certain nerve districts, for instance, in the distribution of the facial (unilateral or bilateral mimetic facial convulsions), or in the muscles of the eye (nystagmus). Unlike the convulsions, the *paralyses* are developed usually in the retrogressive stage, and are of long, even life-long duration. The cranial nerves, particularly the abducens, oculomotorius, and facial, may be thus affected, the paralysis, perhaps, depending mostly upon lesions of the respective n trunks in their course to the base of the skull, as a result e of the surrounding exudation (purulent infiltration of the rilemma), or of the contraction of the hyperplastic conne tissue of the nerve sheath. This explanation is the more sible from the fact that, in the cases which have been care examined, the paralyses showed all the characteristics of *pheric* lesion, and that the nerves mentioned were found a autopsies so frequently and markedly enveloped by the lent exudation that it is remarkable that paralysis of the nial nerves is not more common.

Central paralyses originating from the brain or spinal are not often found (hemiplegia, hemiparesis, paralysis o or both lower extremities), yet it must be borne in mind th severe cases, particularly where there is loss of consciou these symptoms are with difficulty discovered and easily looked.

Aphasia and *anarthria* likewise occur (Leyden).

The *eye* and *ear* have been found to be more frequ affected than the other organs of special sense.

Disturbances of hearing usually begin in the first few The patient complains of pain, humming, and ringing in the then deafness rapidly ensues, usually in both ears. After time hearing is partially or wholly restored, or complete ness results, which, as a rule, is incurable. I have observed symptoms eight times in forty-eight cases.

In a few cases examined by A. Heller, Klebs, and Luca lesions were found to consist sometimes of suppurative infla tion of the labyrinth, with destruction of the membranous rinth, sometimes of catarrhal or purulent inflammation o middle ear. The latter condition is evidently less dange because, as I myself have observed in one case, the perfor of the membrana tympani results in only a moderate deaf The investigations of Heller render it very probable tha inflammation makes its way within the sheath of the aud nerve

The ocular lesions consist partly of mild or severe conju vitis, and opacity and ulceration of the cornea—the latter, have shown, frequently depending upon incomplete closure of the lid from paresis of the orbicularis palpebrarum muscle, in consequence of the cornea being exposed, in the region of the opening, to injury from the air—partly of severe suppurative irido-choroiditis, resulting in detachment of the retina, cloudiness of the lens, and atrophy of the bulb; partly of optic neuritis terminating in atrophy of the nerve. These inflammations, like the auditory lesions, probably depend upon the extension of the inflammatory exudative process to the base of the skull, along the sheath of the optic nerve (Schwalbe).

The marked *chemosis* which sometimes surrounds the cornea like a wall, results, according to Leyden,[1] either from suppurative infiltration of the fatty tissue of the orbit, or from severe panophthalmitis. The paralysis of the ocular muscles, especially the abducens and oculo-motorius, has already been considered. The *pupil* is often normal during the whole course of the disease, and becomes dilated only towards the end; but is frequently contracted at the outset, and dilates first after two or three weeks.

The *digestive organs* early exhibit various derangements. In addition to the vomiting, which has been mentioned as a leading symptom, there are nausea, anorexia, and retraction of the abdomen; less frequently meteorism and constipation; in some cases diarrhœa, and a moist, heavily coated—but only in comatose cases dry—tongue.

The *nutrition* suffers seriously, especially in the severe forms of the disease, on account of the obstinate vomiting, anorexia, sleeplessness, fever, and severe affections of the nervous system. Hence the great emaciation which the corpse presents after a long illness.

Besides icterus, which occurs in a few cases, the *liver* presents no evidences of derangement.

The *spleen* is sometimes moderately enlarged. It is only rarely that the increased size can be discovered either during life or after death, nor are the intermittent cases characterized by this symptom. We have not observed tumors of the spleen so frequently as Wunderlich and Mannkopf have.

---

[1] Virchow's Arch., Bd. 29, p. 199.

Inflammation of the *parotid gland* likewise occurs b rarely.

As regards the *urinary organs*, the quantity of the secreti is often much increased even during high fever.

In one case, a girl eight years of age, we noticed polyu for some six or seven days, in connection with slight albu nuria and moderately high fever. Mannkopf[1] likewise obser marked polyuria in a case similar to ours, and afterwards sug but no albumen. Mosler has described one case of hydru lasting for years in a boy seven years old, as a sequel of cereb spinal meningitis from which he had recovered in his third ye

It is probable that both the polyuria and the melituria neurotic derangements; at least this is the explanation wh was formerly given by Mosler and myself, and yet the relat frequency of the deposits of exudation which we have found the fourth ventricle would seem to demand a more freque occurrence of these symptoms.

The reaction of the urine is generally acid. Exceptiona we find deposits of urates and phosphates, and a small amou of albumen. In comatose cases retention of urine read occurs, and, if the catheter be not used at the proper ti cystitis.

Organs of Circulation.—The characters of the pulse ha already been considered. Pericarditis and endocarditis are ra complications.

The *respiratory organs* very generally show catarrhal aff tions of the bronchial mucous membrane (rarely of the upp air-passages), with secondary atelectasis and broncho-pneum nia. I have already[2] called attention to the fact that the lesions in the inferior and posterior portions of the lungs a found particularly in those patients who for a long time ha had orthotonos, and I believe that this connection may explained by the strong contraction of the spinal-erector mu cles, which renders the proper inspiratory expansion and t forcibly-expiratory compression of these parts of the lungs i possible. On the other hand, it is supposable, although not ve probable, that the broncho-pneumonic areas may sometimes be produced by the gravitation of the fluids used as food into the parts, in consequence of the difficulty of swallowing caused by retraction of the head. Against this view we have the fact that the pneumonic infiltration was absent in those of our cases in which there was merely stiffness of the neck but none of the spinal column, while in ten cases of marked contraction of the whole spine the consolidations were present nine times (broncho-pneumonia eight times, collapse of the lungs on both sides once).

---

[1] L. c., p. 166.

[2] L. c., p. 378 and 444.

I can say, from my own observations, that the longer the marked orthotonos continues during life, the more certainly we find at the autopsy inspissated secretion in the bronchi of the lower lobes, lobular collapse, and lobular pneumonic infiltration.

In the fulminant cases the bronchi and lungs are entirely unaffected.

The *frequency of respiration* is rarely much altered at first, being generally normal. The absence of the well-known loss of rhythm of tuberculous basilar meningitis is quite noticeable. Towards the end, the breathing usually becomes very rapid, and then arythmic, the respirations regularly alternating with respiratory pauses, as is the case in the Cheyne-Stokes symptom. Leyden supposes that this irregularity is due to pressure on the medulla oblongata, or to œdema of the same.

Complications and Sequelæ.

The most frequent complications, those occurring in the lungs, ear, and eye, have been already considered in the analysis of the individual symptoms. It may here be added, that pleurisy, endocarditis, pericarditis, diphtheria of the intestines, parotitis, and purulent effusions into the joints occur as complications of some cases in every epidemic.

The *inflammatory processes in the lungs* are by far the most serious of these intercurrent affections, yet according to our observations they very readily undergo resolution if they are developed as secondary broncho-pneumonias.

Much more serious, however, is the occurrence of *croupous* *pneumonia* during epidemic meningitis, or, on the other han the development of the latter in the course of croupous pne monia.

The latter coincidence, viz., the *occurrence of cerebro-spin meningitis*, during an epidemic, *as a complication of other ac diseases*, particularly of the true croupous pneumonia, is t more remarkable from the rarity with which this complicatior observed in ordinary times.

The older literature in regard to epidemic meningitis gives little information on this subject. All the more valuable, the fore, are the special investigations on this point by Immerma and Heller,[1] in the Erlangen epidemic, and by Maurer.[2] It pears from their reports that the combination of croupous pn monia and cerebro-spinal meningitis was most frequent after t termination of the epidemic; as if the infectious poison h then lost its violence, and was able to resume its activity o when aided by the force of other diseases.

The cases collected by Immermann and Heller, and the obs vations at Erlangen, published by Maurer and myself, show t following results for 1866–72:

Meningitis as complic. croup. pneumonia, 14 times.
" " scarlatinal nephritis 1 time.
15 times.

Of these cases, six were children in their first year, while in t epidemics of 1864–66 no nursing infants were attacked.

The symptoms of this form of cerebro-spinal meningitis of so slightly marked a character that, in many of the ca observed by us (see Immermann and Heller), the diagnosis w uncertain, or could not be made at all. Generally, after t pneumonia had lasted for one or more days, the headache b came worse, and other severe characteristic cerebral sympto appeared: delirium, convulsions, coma, vomiting, stiffness the neck and spine, and contraction of the pupils. It must borne in mind, however, that the contraction of the neck is oft absent, even in severe meningitis. The difficulty of diagnosis is greatest in children under one year of age, in whom convulsions, especially when occurring during the first few days, slight stiffness of the neck, somnolence, and coma are not characteristic indications, and in whom also the existence of headache cannot be ascertained with certainty.

---

[1] Pneumonie und Meningitis, Deutsches Arch. f. klin. Med., 1869, Bd. V, p 1 et s

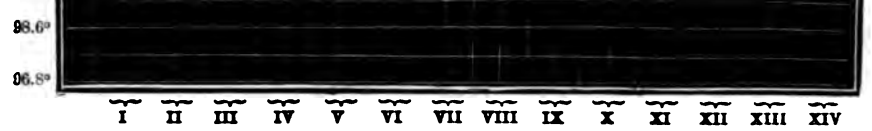
[2] Pneumonie und Meningitis bei Kindern, Deutsches Arch. f. klin. Med., 1874, B XIV, p. 47.

Maurer calls attention to the great importance of examining in young infants the *great fontanelle, which is found arched and tense*, as a result of the increased intracranial pressure from the meningeal exudation and œdema.[1] This symptom in young children is very significant when it occurs in the course of a pneumonia and in connection with convulsions, coma, retardation of the respiration, and stiffness of the neck.

The prognostic indication of this complication is very serious, since almost all the cases terminate fatally.

I give briefly, with the temperature curve, an example of this complication.

A. Wünsch, aged thirty-six years, a Prussian militiaman of delicate constitution, was taken sick with a chill, while at work, on the evening of August 22, 1866, at Erlangen, and was received into the medical clinic on the 23d. On the 24th a pneumonia of the upper lobe, with high fever, was discovered. Unusually severe headache, also vertigo. On the 25th, headache more intense, great restlessness, delirium, and hallucinations. Mind clearer after the application of ice-bladders. On the 27th and 28th the pneumonia extended to the inferior and midde lobes. Mod-



---

[1] In one of *Maurer's* cases the arching and tension were so great, that neither the edges of the bones nor the pulsation of the brain could be felt.

erate headache, somnolence, slight delirium. During the night of the 28th and 29t active delirium; somnolence in the morning. Fever still high (see diagram). Aft noon, August 30, continued delirium; at night great restlessness, wanders about t room, hallucination. Evening, August 31, conscious, vomiting of milk, compla of headache. Evening, September 1, active delirium, loud expressions of pain w patient rises, spine somewhat stiff, meteorism. During the night of September 2 actively delirious, wanders about the room. Evening, September 3, muttering d rium; on rising, complains much of pain in the loins and back. September extreme collapse, coma. Death at noon, September 5.

Autopsy (twenty-four hours after death).—Croupous pneumonia on the ri side, suppurative cerebro-spinal meningitis, œdema of the lungs, bronchial cata enlargement of the spleen and kidneys.

The more important of the sequelæ are *deafness*, *derang ments of vision*, *chronic hydrocephalus*, and *chronic meningit* with the consequent impairment of intelligence, and lesions *motility* in the form of paralyses and pareses.

1. The *auditory lesions* may occur either in the middle e or in the labyrinth. The true inflammations of the middle e usually lead to perforation of the membrana tympani, and pr duce more or less deafness. In one case[1] which came under o observation, the otitis began with severe pain on the twenty-fi day. On the thirty-sixth day the pus discharged through t membrana tympani, cicatrization of the perforation took pla and slight deafness in the right ear resulted.

Suppuration in the labyrinth is far more serious, affe generally both ears, and usually ends in complete deafness.

In regard to the anatomical conditions which cause the frequently occurring sequelæ, it may easily be conceived th the suppurative exudation produces some effect upon the str of the auditory nerve at its origin in the fourth ventricle, or up the trunk of the nerve. And yet I have often found the floor the fourth ventricle macerated by pus, and the auditory nerv together with the facial nerves, completely surrounded by t purulent exudation, without the occurrence of deafness duri life; and on the other hand, in cases of marked deafness duri life, several observers have found very important changes in t middle and internal ear, such as purulent and adhesive infla mation in the tympanum, and suppurative inflammation in t labyrinth with ecchymoses (Heller, Merkel, Klebs). The auditory nerve was found surrounded and much infiltrated by pus in the meatus auditorius internus. We must, consequently, suppose, either that the inflammation creeps along the sheath of the auditory nerve, and enters the labyrinth, or that the lesions in the tympanum and labyrinth are developed simultaneously with the inflammation of the pia mater, as co-effects of one and the same destructive cause. The former supposition is regarded by Heller as the more probable. At the same time, it is remarkable that the facial nerve is so resistent to the purulent infiltration, as has been noticed by Heller in two cases. This is undoubtedly the explanation of the fact that the function of the facial always remains intact; at least, I have observed this to be the case in all the patients I have seen with complete deafness.

---

[1] L. c., page 390.

The frequency of impairment of hearing is much greater, according to my observations, than one would suppose from the statements of writers. I was much surprised at the large number of completely deaf children who were brought to me for examination from the rural districts of central and upper Franconia, during the first few years after the termination of the epidemic.

In children under one year of age complete deafness results in *deaf-muteism*, if they have not previously learned to talk; in the second, third, and subsequent years, in the loss of articulate speech, even if the child had fully acquired the power. In the latter case, several months after the occurrence of deafness, the speech can be understood with difficulty, and after a year or more, becomes quite inarticulate and unintelligible. Such children should be placed under the instruction for deaf-mutes, if their speech is to be of any use to them in later life. Forget, Lindström, and others, have noticed that deaf-muteism is a rare result of the meningitis, even in those cases in which hearing is completely lost. Hirsch has called attention to the fact that impairment of articulation, and even aphasia may occur at the same time with the deafness. In those cases of which Hirsch and Leyden have observed examples, the loss of speech is not the result of the deafness, but is a co-effect of the meningitis. I cannot remember any such cases; but I am willing to grant the possibility of such an acute origin of impaired articulation fro the meningitis, perhaps by means of œdema or hemorrhages the spinal cord. In the numerous instances which I ha observed, the articulation was good for a short time after t meningitis, and became worse gradually.

I am indebted to Dr. Roth for some very interesting statements in regard to pupils of the deaf-mute institution in Bamberg. This charity is intended only the deaf-mutes from the district of upper Franconia. The number of pupils April, 1874, was 42 (20 boys, 22 girls). *All the pupils had become deaf-mutes f epidemic meningitis.* Thirty-eight of them came from the rural districts, and o four from Bamberg, although the epidemic in Bamberg had been very severe.

I am also indebted to Dr. Merkel for similar accounts of the deaf-mute inst tion in Nuremberg. The number of deaf-mutes from meningitis was 22 (10 b 12 girls), out of a total of 32 pupils.

2. The more important and frequent sequelæ of the *affectio of the eye* are opacities of the cornea, posterior synechia, etc., results of iritis; amaurosis from choroiditis with detachment the retina, and finally optic neuritis with secondary atrophy the nerve. These severe lesions depend either upon an ext sion of the inflammation along the sheath of the optic nerve. upon a localization of the suppurative inflammation in the e and brain at the same time (Rudnew).

3. *Permanent affections of the brain, the spinal cord, a their membranes* are by no means rare. In my opinion, by far t most frequent of these sequelæ is *chronic hydrocephalus.* least I have seen a very large number of hydrocephalic patien and have made autopsies on some of them, both during t course of the epidemic and in subsequent years. These exan nations show that the successive changes in the central orga take place in very nearly the following order: During t second week the meningeal exudation, which has hitherto be little changed, or perhaps somewhat thickened, undergoes fat degeneration of the cells and fibrine, and is thus slowly or rapid absorbed, or ultimately shrinks into caseous matter, if absor tion does not occur; the connective tissue of the arachnoid a pia mater proliferates, the hyperæmia of the substance of t brain disappears, and the purulent effusion in the ventric increases. From the twenty-seventh to the thirtieth week t

arachnoid and pia mater exhibit a pulpy hyperplasia or already a cicatricial thickening; the caseous remains of the meningeal exudation are still more shrunken; the ventricular effusion has become more moderate in amount, but quite clear, owing to the inspissation of the cellular elements into small caseous flakes on the dependent parts of the ventricle. The earlier hyperæmia of the brain is completely gone, the brain is anæmic, even œdematous, the ependyma of the ventricles thickened and distinctly granulated, and the choroid plexus bloodless. Unless the hydrocephalic effusion be moderate, the brain substance is atrophied sometimes to a very considerable degree. In a boy two years of age we found the medullary and cortical layers of the cerebrum together only seven to nine and a half lines in thickness, while the central ganglia were much flattened.

It almost appears as if the increase of the ventricular effusion were due to the shrinking and thickening of the pia mater; at least this supposition is rendered probable by the interval of apparently progressive convalescence which usually occurs between the acute stage of the meningitis and the appearance of the hydrocephalic symptoms.

The *symptoms of secondary hydrocephalus* consist of paroxysms of severe headache, pains in the neck and extremities, with vomiting, loss of consciousness, convulsions, and involuntary discharges of fæces and urine. These attacks recur either in rapid succession and tolerably regular type, or with intervals which sometimes last for weeks, and during which the activity of the mind and senses may be unimpaired, the appetite excellent, the sleep quiet, and the patient in good spirits and gaining strength. If the intermissions be long continued, we easily fall into the error of supposing that the disease has taken a favorable turn; but a new attack dispels the illusion. Moreover, the condition of the patient during the intermissions is generally not so favorable as to lead to such a mistake in prognosis; there are still to be found the general hyperæsthesia and increased reflex excitability, the slight contractions or pareses in the extremities, psychical disturbances, and other symptoms.

Of far greater importance is the question, *whether and to what extent these hydrocephalic effusions are capable of retro-*

gressive changes; in other words, whether recovery from secon ary hydrocephalus is ever possible.

The prognosis of this condition is certainly in the high degree hopeless; yet I have seen some cases in which a comple and others in which an incomplete, recovery took place. Of first class of cases I have already published one instance (Arch klin. Med., Bd. 1, page 389); of the latter, the following i very instructive case, in which the autopsy was made seven ye after the attack of meningitis.[1]

Joseph Klaussner, aged 19 years, son of a farmer at Rockenhof, near Erlan passed through an attack of epidemic meningitis in his twelfth year (1865), a same time with several of his sisters (one of whom died). He was confined t bed twenty-eight weeks. The most prominent symptoms were stiffness of the trismus, very severe pain in the extremities, especially in the fingers, so th wished to pull them off, and impairment of hearing. In consequence of his e sive weakness and emaciation, the convalescence was very tedious; yet he reco completely. There was left, however, great feebleness of memory. He fo everything which he had learned before his illness. Headache, especially on st ing; afterwards he suffered from weakness of the eyes, and remained stationar his growth.

In January, 1872, he had a discharge from the ears, a weeping eruption on head, and enlarged glands in various parts of the body. The left side of the was numb; yet the patient regularly walked the long distance to Erlangen in hours.

Patient states that at the beginning of February he had for fourteen days p in the neck and difficulty in swallowing (diphtheria?), yet he was not confine bed.

At the end of March the right side of the body was numb (with the exceptio the leg), and his speech had been of a nasal character for four weeks. On ea and drinking, the food passes into the larynx and naso-pharyngeal space. staggering, now and then vertigo, enormous salivation, near and distant vi defective.

Present State.—A small man, of stupid appearance, deficient intellige childish frame and voice, yet with well-developed pubic hairs. Paralysis of half of the soft palate, which presents no reaction to the farado-electric, increased sensibility to the galvanic current.

After several local applications of electricity to the paralyzed muscles, nasal character of the voice and the difficulty of swallowing disappeared. improved; right arm still numb.

---

[1] For the notes of this case, and for the opportunity of attending the autopsy, I indebted to the kindness of Dr. Maurer, of Erlangen.

On April 1 patient returned from a visit to the country apparently well; was taken ill early in the day, on April 2, with high fever, and died suddenly about one o'clock in the afternoon.

Autopsy.—Marked hydrocephalus, with thickening of the ependyma. The central canal of the spinal cord was dilated. Remains of cerebro-spinal meningitis, in the form of opacity and callous thickening of the pia mater in different places.

Double pleuropneumonia, emphysema, and œdema of the lungs. Old bronchiectases. Moderate hypertrophy of the heart. Hyperæmia of the liver and kidneys.

The chronic hydrocephalus, with thickening of the ependyma and hyperplasia of the arachnoid and pia mater, must properly be regarded as a sequel of the meningitis. At the same time it is questionable whether the impairment of intelligence and of bodily development is likewise and exclusively due to the reaction of the hydrocephalus and meningeal shrinking upon the central organs of intelligence and upon the trophic functions.

So many cases of secondary hydrocephalus similar to mine have been observed within the last few years, that the disease can no longer be regarded as a rare sequel of meningitis. Reports of such cases have been made by Merkel, Böhmer, Bonsaing, Pimser, and others. v. Lindwurm observed a very instructive case of this kind at the beginning of the year 1874, in his clinic in Munich.

A baker, 18 years old, previously always healthy, was taken sick Jan. 12, 1874, with repeated vomiting and severe headache; on the next day, lumbar pains, double vision, contracted pupils, stiffness of the neck, herpes; temperature between 102.2° and 104°; pulse 100–120; urine abundant, without albumen or sugar.

After January 18, gradual decrease of all the symptoms. Towards the end of the mouth, returns of the headache, involuntary stools, imbecility. On the 29th, two attacks of vomiting, some diarrhœa, pupils dilated, temperature about normal. After this time, intermissions of one to three days' duration alternated with periods of exacerbation, during which there were vomiting, headache, dilatation of the pupils, somnolence, and general malaise with pain. On the favorable days the appetite was good, and there was no fever. On March 4 patient felt so well that he requested to be discharged; in the evening facial paralysis was noticed, and the bowels moved involuntarily. Abscess in the glutæi muscles. At the end of March, after long-continued alternation between improvement and symptoms of pressure upon the brain, the patient was extremely emaciated, exhibited marked decubitus and slight dulness over the right apex anteriorly. Pulse varied between 75 and 100.

After the beginning of April the pulse continued constantly above 120. Abscess in the nipples. Dulness over right apex increased, without bronchial breathin pneumonic sputa. Decubitus very marked, emaciation and extreme prostration.

Death on April 20.

Autopsy.—Convolutions of the brain much flattened. A large quantity of ser in the subarachnoid space, particularly at the base. Ventricles enormously dila and filled with turbid serum. Substance of brain anæmic. Recent desquamat pneumonia of the upper lobe of the right lung.

Feebleness of mind and memory, aphasia, and anarthria often observed as sequelæ, but generally the prognosis is fav able, as they usually disappear within a few months.

Paralysis of *single extremities* or *nerves*, especially of cranial nerves, or general motor weakness, are not very infrequ complications or sequelæ, and seem to depend upon lesions eit in the brain or in the spinal cord. Hitherto there have b found in some cases only disseminated encephalitis (Klebs), punctate hemorrhages in the spinal cord, and an extension the inflammation to the cord (Mannkopf). Leyden[1] saw, i boy five years of age, a *myelitis* which had resulted from ce bro-spinal meningitis, and which was much improved by to treatment. (The final result is unknown, as the patient left hospital too soon.)

Tourdes, de Renzi, Leyden, and others state that the progno in these paralyses is not unfavorable, as recovery takes place most of the cases within a few months.

Diagnosis.

The diagnosis of epidemic meningitis usually presents difficulties, if the disease occur primarily and during the pre lence of an epidemic. The acute onset, with or without prod mata, the symptoms of violent disturbance of the cerebro-spi system, especially the furious headache, the rachialgia, vomiting, the contraction of the cervical muscles, afterwards orthotonos, the general hyperæsthesia, the alternation of so nolence and delirium, the eruptions, especially herpes faciali all these are sufficiently characteristic symptoms, even for who had never before seen the disease. The occurrence, afte few days, of irregularity of temperature and pulse, of exacerbations, etc., renders the diagnosis certain.

---

[1] L. c., p. 430.

The diagnosis is difficult when,

1st, It concerns isolated cases occurring either beyond the limits or at the beginning of an epidemic, particularly cases of little children; or,

2d, When the epidemic meningitis occurs as a complication of other acute diseases, especially croupous pneumonia.

In the first case, the distinction of *sporadic cerebro-spinal meningitis* from the epidemic form is rendered easy by the great rarity of the former, as well as by the fact that the spinal symptoms are not well marked, nor does it seem to run a hyperacute course. Moreover, the spinal symptoms, hyperæsthesia, stiffness of the spine, with rachialgia, and the pains in the extremities, are often entirely absent, and the contraction of the neck is less than it is in epidemic meningitis.

In *tuberculous basilar meningitis* there may be considerable stiffness of the neck, and similar headache, loss of consciousness, restlessness, with irregularity and moderate height of the fever; but the tuberculous nature of the affection may be distinguished by the long-continued prodromal period—which is very seldom absent—the less violent onset, the more protracted course, the marked remissions, the very slow pulse, the irregularity of the respiration, the absence of eruptions; finally, by the evident hereditary tendency to tuberculosis, as well as the history of scrofulous and phthisical affections. In some cases, especially in children, the differential diagnosis may present great difficulties.

So also under some circumstances, and at the outset of the disease, it may be simply impossible to distinguish the affection from *typhoid fever*. Leyden has called special attention to the fact that the severe headache, vomiting, lumbar pains, sleeplessness, roseola, petechiæ, enlargements of the spleen, indeed, even the lowered temperature, stiffness of the neck, and hyperæsthesia of the extremities may occur in typhus, and mentions in this connection the doubts which were for some time entertained on account of the presence of these symptoms in a number of cases at the outbreak of typhus fever in the army besieging Paris (autumn of 1870).

A positive diagnosis of meningitis may be made, if, after repeated observations, we find the characteristic course of the fever, a contracted condition of the spleen or absence of enlargement, and the occurrence of herpes facialis—a very rare symptom in typhus.

The recognition of epidemic meningitis—when it arises as a *complication of croupous pneumonia and other acute diseases*—we have seen it in acute nephritis, pleuritis, tonsillitis, etc.—presents much greater difficulties, especially during the first years of life, when this complication is most apt to happen. It appears from the observations of Maurer that the diagnosis may be made from the occurrence of *coma* towards the middle or end of the normal course of pneumonia, with *convulsions* which follow immediately, and recur frequently until death ensues; from the *retardation of the respiration* during the pneumonia, and from the *arching of the great fontanelle.* In adults, so far as I have observed, more importance is to be ascribed to the apparently causeless occurrence of headache, or to its sudden increase when it is already present, to the painful stiffness of the cervical vertebræ, the hyperæsthesia, restlessness, active delirium, and coma. I am convinced, from the observation of cases in the Erlangen clinic and polyclinic,[1] that the high temperatures and frequent respiration of pneumonia may continue unchanged, notwithstanding the meningitis, and moreover, that the alterations of the pupil, the vomiting, the eruptions, and even the stiffness of the neck may be absent. So also the majority of the cardinal symptoms of meningitis may be wanting, and the diagnosis may thus be made doubtful for some time.

Course, Duration, and Terminations.

As we have seen in the description of the general symptoms, the course of the disease is very variable. The hyperacute and the abortive cases run the most rapid course (one and a half to five days), while those of moderate severity begin to convalesce after one or two weeks, but sometimes last for months. When complications or sequelæ arise, the whole duration may be six months or more.

---

[1] See Immermann and Heller, l. c.

In the fulminant cases death is the rule; in the moderately severe attacks it is still comparatively frequent, and even in the mild cases it sometimes occurs as a result of the complications or sequelæ. The rate of mortality varies very much according to the intensity of the epidemic; in the mildest epidemics, 30 per cent., in the most severe, over 70 per cent. It appears at least very probable that the excessive percentage (70 to 80 per cent.) which occurred during the French epidemics of the decade following 1840, was due to immoderately antiphlogistic treatment.

The mortality will average about 40 per cent.

Prognosis.

In individual cases the prognosis can never be made with certainty. To be sure, we may say in a general way that in the fulminant cases the patients die, while in the mild and abortive ones they recover; but the exceptions are so numerous that we can be guided in our prognosis only by probabilities. In the majority of severe and moderately severe cases, any prediction of the result must be withheld for the first few days, and we must wait for further developments.

The following conditions are generally to be regarded as of unfavorable import: infancy and old age, unusual severity of the symptoms of excitement, and the early appearance of those of depression, return of the vomiting, intense cephalalgia, and loss of consciousness after apparent improvement, continuous coma, convulsions, and irregularity of the respiration.

A favorable prognosis may be made in the moderately severe and mild cases, when all the symptoms uniformly decline in the first and second weeks, and convalescence takes place during the third or fourth weeks without the development of important complications or sequelæ.

TREATMENT.

In our ignorance of the etiological conditions of the disease, *prophylactic measures* can be recommended only in a general selves to the study of the immediate effect upon the subjective and objective condition of the patients, and to the duration of that effect. Then let conclusions be drawn from the largest possible number of similar cases.

Antiphlogistic treatment, by means of local *abstractions of blood*, leeches behind the ears, and ice-bags, is very efficacious. We have found but few patients who did not praise the application of ice to the head. That many patients repeatedly pull off the ice-bags while they are comatose or delirious, is no evidence that the cold is not well borne. The ice-bags are to be left upon the neck and back so long as the patient is comfortable, and this is often a long time. One old woman bore the application uninterruptedly for twenty days and nights, and afterwards, at intervals, for three weeks longer, for the relief of recurrent headaches.

The cold almost always relieves the severity of the headache, and secures rest and sleep. It is best applied by means of large rubber bags. Every time ice is introduced, all the air should be pressed out before the cork is inserted, in order that the bag may fit the scalp smoothly.

Cold should also be applied to the neck and back. For the spine, Chapman's long ice-bags are most suitable, which are divided into three adjacent compartments for the ice, and can be fastened around the body by means of tapes. I have had rubber bags made twenty-three and a half to twenty-seven and a half inches in length, for the purpose of covering the whole length of the spine.[1]

Abstraction of blood is best made by leeches to the temples or mastoid processes, and by the application of wet cups to the neck or along the spine. General *blood-letting* has nowadays few advocates among those who have seen most of the disease. The effect of this measure in subduing the cerebral symptoms is comparatively slight and transitory, while a large venesection produces those evil results which are found to occur in all infectious diseases, such as collapse, weakness of the heart, etc. It is still to be considered, however, whether in hyperacute cas which all treatment has hitherto been unavailing, a rapid copious withdrawal of blood from a vein may not avert least postpone the fatal termination. The acute congesti the brain and its membranes, if not the only, is at leas important cause of the unfavorable course of these c Leyden, who favors venesection in such attacks, main moreover, that under proper circumstances general blood-l is indicated by the excessive restlessness of the patient, renders the application of leeches almost impossible.

---

[1] These long ice-bags with several compartments are well adapted for the application of ice to the thorax in double pneumonia, to the abdomen, or to the extremities. Unfortunately they are very expensive.

Local abstractions of blood, even when frequently rep produce almost always and immediately a very beneficial They are most suitable at the beginning of the exacerbati inflammation and fever. The diminution of the intra-ce hyperæmia is shown by the relief of the headache, jactit and delirium, and by the return of consciousness.

Mercury, in the form of mercurial ointment or calome formerly and is still much used for the purpose of pr ing the extension of the meningeal inflammation and exud I have used it in most of the cases from the beginning of t ease. In adults about thirty grains, in children fifteen to t two grains of mercurial ointment were rubbed into the skin daily (the place being regularly changed). Calomel was always given in large doses with jalap, for the purpose of freely upon the bowels, and thus producing an active dete tion to the intestinal mucous membrane.

For the purpose of preventing stomatitis, the mout washed every half hour, after the commencement of the curial treatment, with pure water and with a solution of ch of potassa, twenty-five grains to the ounce. Althoug drachm of mercurial ointment, and on an average about t grains of calomel were daily used in the adult cases, the local ment of the mouth prevented the occurrence of salivation except a few instances, and in these the symptom gave but trouble.

When mercury is employed in connection with other dies, it is difficult to ascertain its share in the common effec even when used alone its efficacy is by no means clearly lished. Still, Leyden believes that it does check the inflammation and the exudative process. Its employment may be the more recommended as we possess no remedy which is more effectual.

The opinion of the older writers, that the occurrence of mercurial stomatitis is a favorable indication, has not been confirmed by my experience ; indeed, in most of the protracted cases death was preceded by a mild stomatitis.

The *antipyretic treatment* by cold baths and large doses of quinine is indicated in but few cases, as there is generally only a moderate fever, and it is very rare that the fatal result is due to a high temperature. Besides, as Leyden has insisted, patients with meningitis usually bear the baths badly, on account of the resulting depression and the pains caused by the unavoidable movements of the spine, etc. Cold affusions are also not well tolerated, and should be used only in the very acute cases, and when the patient is soporose.

Quinine may be employed as an antipyretic in doses of fifteen to thirty grains, in those rare cases in which the temperature ranges very high. It is entirely useless against the exacerbations of the intermittent form.

Narcotics play an important part in the whole course of the treatment. The majority of recent writers confirm the happy effects which Chauffard has noticed and praised so highly.

The violent headache, restlessness, and sleeplessness are sufficient indications for the administration of opium and its alkaloids, of which *morphine*, and particularly by hypodermic injection, acts the most promptly. If we wish to secure a quick and certain effect, we may use quite large doses (one-third to one-half grain in adults) without fearing any injurious consequences. *Smaller doses* administered every one or two hours, when tolerated by the stomach, appear to be of great service in relieving the incessant jactitation. For the exacerbations of the cephalalgia it is better to give a larger dose of morphine by injection. If the latter be used immediately after a local blood-letting, the most restless delirium gives place to quiet and sleep, which often last for seven or eight hours. *Morphine may be regarded as one of the most indispensable remedies in the treatment of epidemic meningitis.*

As useful palliative measures, we may use ether droppe sprayed upon the back of the head and neck, and friction chloroform liniment. Inhalations of chloroform or ether, large doses of chloral are recommended by many writers, are, at all events, worth the trial.

In the later period of the disease, especially when the c has been protracted, *iodide of potassium* is of service in p cing *absorption of the exudation.* Traube, Leyden, and m have seen good results from this treatment, convalescence t place with unusual rapidity. In chronic hydrocephalus I found it to be entirely useless.

The hydrocephalus unfortunately withstands all other ment, whether external or internal. Applications of cold give relief in many cases, but the improvement is always temporary. Counter-irritants to the neck and scalp, se etc., are without apparent effect.

Derangements of hearing and vision are, of course, the o of special therapeutic attention. In severe cases, either of ness or amaurosis, but little success can be expected. Th vanic treatment of deafness following meningitis is illusive I herewith record my warning. In cases of complete dea this treatment is absolutely useless, and no one has eve produced electric auditory sensations.

The *diet* must naturally be regulated according to the dition of the fever, etc. After the disappearance of the f a nutritious, but fluid, diet should be adopted early, in vi the fact, which experience has shown, that a disproportion great emaciation and prostration commonly occur in meni after the patient has been confined to bed for but a short ti

The other sequelæ, such as general motor weakness, va pareses, etc., may be treated by the use of the saline bat Kreuznach and Reichenhall, or by the hot baths of Wiesb Wildbad, Ragatz, and Gastein.

INDEX.

AARESTRUP, 100.
Abeille, 81.
Abelin, 44, 60, 99, 123
Ackermann, 50.
Aconite in erysipelas, 478.
Age in epidemic cerebro-spinal meningitis, 698; in erysipelas, 431, 477; in hay fever, 542; in influenza, 522: in malarial diseases, 573; in measles, 48; in miliary fever, 493; in rubeola, 136; in scarlatina, 178, 294; in small-pox, 326, 393; in varicella, 8.
Albers, 39, 222.
Albu, 311.
Alison, Scott, 178.
Amiel, 480.
Anatomy of the skin in small-pox, 380.
Angerhausen, 467.
Anstie, 469.
Aran, 398.
Arlt, 434, 456, 469.
Armand, 665.
Arnold, 133.
Arnott, 459.
Arrigoni, 282.
Arsenic in malarial diseases, 671.
Ascites in scarlatina, 257.
Astringents in erysipelas, 480.
Aufrecht, 482.
Auspitz, 384.
Avé-Lallemand, 509.
Avery, 443, 466.

BACTERIA in erysipelas, 445.
Baginksky. 247, 305.
Baikee, 657.
Baillie, 43.
Balfour, 479, 657.
Ballonius, 36.
Ballot, 510.
Barat, 572.
Barbillier, 44.
Barclay, 523.
Barella, 665.
Bärensprung, 382, 631.
Barker, 196.
Barnatzik, 663.
Barrant, 672.
Bartels, 99, 101, 103, 123, 124.
Barthez, 47, 91, 105, 204, 274, 492.
Basch, 384.
Bashan, 314.
Bastard, 503.
Battersey, 43, 101.
Baxa, 657.
Baxter, 673.
Bayer, 413, 458, 459, 461.
Bazin, 499.
Bechi, 571.
Beck, 496.
Becquerel, 81.
Beger, 102.
Béhier, 43, 460.
Behr, 44.
Behrend, 165.
Bell, 165.
Bell, Hamilton, 479.
Beneke, 450.
Bennett, 307, 443.
Bentley, 101.
Bernatzik, 665.
Berndt, 40, 659.
Bernutz, 433.
Beroaldi, 496.
Bertherend, 670.
Berton, 189.
Betz, 219, 233, 266, 274, 486.
Bicker, 214.
Bidenkap, 44.
Bierbaum, 42, 43, 47, 600, 610.
Biermer, 223, 228, 522, 524, 525, 526, 530, 534, 538.
Biesiadecki, 444, 452, 455.
Biett, 496.
Billard, 474.
Billroth, 447, 451, 477.
Binz, 121, 479, 664, 667.
Bird, Hinckes, 430, 478.
Blache, 431.
Blake, 479.
Blackett, 432.
Blackley, 540.
Blanckaert, 91.

Blandin, 456.
Blass, 459.
Blaxall, 566, 587.
Blood in erysipelas, 444; in malarial diseases, 624, 633; in miliary fever, 496; in scarlatina, 233.
Boeck, 27.
Bohn, 253.
Bokai, 172.
Bonaventura, 664.
Bones in scarlatina, 232.
Bonfigli, 481.
Bonsaing, 698.
Borchard, 496.
Borelli, 602.
Borgien, 481.
Bordeu, 426.
Bostock, 540.
Böttiger, 44.
Bouchut, 206, 474.
Boudin, 580.
Bourgeois, 496.
Bousquet, 412.
Boussingault, 571.
Bouvier, 667.
Boxa, 586.
Boyer, 426.
Brain in epidemic cerebro-spinal meningitis, 705, 717, 726; in erysipelas, 462; in scarlatina, 236; in small-pox, 387.
Braun, 102, 211, 588.
Bressler, 44, 101.
Bright, 480.
Briquet, 665.
Broadbent, 434.
Brochu, 458.
Bromfield, 426.
Bronchitis in measles, 92; in scarlatina, 222; in small-pox, 379.
Brotherston, 311.
Brown, 81, 122, 125, 696.
Brückmann, 44, 45.
Brunzlow, 43.
Brylon, David, 507.
Bryson, 657.
Buch, 349.
Buet, 536.
Buffalini, 39, 580.
Bullock, 478.
Burdel, 641, 670.
Busch, 131, 469.

CAISNE, 672.
Calmeil, 424.
Calvert, 672.
Campbell, 435, 479.
Canstan, 670.
Canstatt, 12, 21, 27, 132, 204, 210, 226, 426, 47
Carpenter, 178, 198.
Carroll, 102.
Causit, 101.
Cazenave, 426, 469.
Cellulitis of neck in scarlatina, 217, 225.
Cerebro-spinal meningitis, epid
683; *bibliography*, 683: definition of the
687; *history*, 687; *etiology*, 690; relatio
other infectious diseases, 691; influence of
and weather, 693; age, 693; hygienic inf
695; sex, 696; the question of contagi
696; *pathology*, 697; general description
disease, 697; meningitis cerebro-spinalis s
699; meningitis cerebro-spinalis abortiv
meningitis epidemica intermittens, 702;
epidemic meningitis, 703; *pathological a*
703; emaciation, 703; the skin, 704; the
um and membranes of the brain, 704; me
of the spinal cord, 705; the brain substan
substance of spinal cord, 706; internal
706; organs of special sense, 707; *ana*
symptoms, 707; headache, 707; vertig
vomiting, 708; contraction of muscles
and spine, 708; trismus, 710; pain in th
710; hyperæsthesia, 710; cutaneous lesio
temperature of the body, 712; nervous sy
717; the ear and eye, 718; digestive orga
urinary organs, 720; organs of circulatio
respiratory organs, 720; *complications*
quelæ, 721; *diagnosis*, 730; *course, d*
and terminations, 732; mortality, 733; *pr*
733; *treatment*, 733; antiphlogistic mea
mercury, 736; antipyretic treatment, 73
nine and narcotics, 737; iodide of potassiu
diet, 738.
Champouillon, 469.
Chauffard, 690.
Chauveau, 381.
Chevrey, 664.
Chinnock, 44.
Christie, 512, 514.
Chomel, 74, 431.
Clarus, 51.
Cohn, F., 381.
Colchicum in erysipelas, 478.
Coley, 102.
Colin, 456, 462, 586, 657.
Collin, 133.
Condamine, 332.
Contagiousness of epidemic cerebro-spinal men
696; of erysipelas, 432; of dengue, 509; o
enza, 523.
Conté, 480.
Copeman, 173.
Copland, 478, 481, 534.

Cornaz, 121.
Cornil, 434, 458, 475.
Corson, 173, 567.
Coulson, 104.
Coural, 490, 497, 503.
Cox, 478.
Coze, 39, 162.
Cramer, 47.
Crato, 524.
Cremen, 177, 178, 278.
Creutzer, 480.
Cullen, 40, 426.
Currie, 120, 673.
Curschmann, 672; on small-pox, 317.
Czakert, 10, 25.

D'ALVES, 185.
Dance, 183.
Dandy Fever, see Dengue.
Danielssen, 281.
Daudé, 433, 438, 443.
Dean, 480.
Declat, 672.
Deiters, 274.
Delpech, 13.
Van Dembusch, 526.
Dengue, 505; *bibliography*, 505; *history and etiology*, 507; *symptomatology*, 510; incubation, 510; fever, 510; the second paroxysm, 511; the joints, 511; the eruption, 511; the mucous membrane, 512; lymphatic glands, 512; the urine, 512; the heart, 513; respiratory organs, 513; convulsions, 513; prognosis, 513; duration of the disease, 513; relapses, 513; treatment, 514.
Derblich, 643.
Després, 462, 466, 469.
Desquamation in miliary fever, 499.
Desiccation, stage of, in small-pox, 363, 369.
Diagnosis of epidemic cerebro-spinal meningitis, 731; of erysipelas, 470; of hay fever, 551; of influenza, 535; of malarial diseases, 652; of measles, 106; of miliary fever, 500; of scarlatina, 284; of small-pox, 388; of varicella, 22.
Diemerbroek, 6, 337.
van Dieren, 42.
Digitalis in scarlatina, 306.
Diphtheria in measles, 99; in scarlatina, 217; in small-pox, 380.
D'Nais, 133.
Dobson, 480.
Doepp, 436.
Doublet, 475.
Drake, 46.
Dropsy in scarlatina, 259.
von Düben, 44.
Duboué, 643.
Dubun, 495, 496.
Duchek, 581, 625, 627.
Dujardin-Beaumetz, 459.
Dumas, 490.
Duncome, 165.
Dunkley, 511.
Dunlop, 133, 140.
Duval, 277.
Dusével, 96.
Dyrsen, 42.

EAR in epidemic cerebro-spinal meningitis, 707, 718, 724; in measles, 95; in scarlatina, 221, 277; in small-pox, 379.
Edwards, 78.
Eichorn, 332.
Eimer, 397.
Eisalt, 44.
Eisenlohr, 672.
Eisenmann, 73, 103, 503.
Eisenschitz, 248.
Emmert, 62.
Emminghaus, 133, 136, 143, 144, 147.
Engel, 391.
Epting, 253.
Erickson, 96.
Erismann, 321, 335.
Eruption in epidemic cerebro-spinal meningitis, 711; in dengue, 511; in measles, 78; in miliary fever, 498; in rubeola, 143; in scarlatina, 441; in small-pox, 355; in varicella, 16.
Erysipelas, 419; *bibliography*, 419; *definition*, 423; *history*, 423; *etiology*, 425; sex, 430; age, 431; occupation, 431; season of the year, 431; exciting causes, 432; contagion, 432; transportation of the disease, 433; infection from the cadaver, 435; stage of disease when infection occurs, 435; resistance to disinfectants, 435; inoculability, 436; by the use of instruments, 436; pyæmic infection, 438; influence of crowding of patients, 439; pollution of soil, 439; defective pipe drainage, 440; *pathology*, 441; symptoms, 441; course of the skin affection, 441; temperature and pulse, 342; the mucous membranes, 442; angina, 449; *anatomical changes*, 443; mucous membranes, 443; congestion and inflammation of lungs, 443; enteritis, 443; the spleen, 444; the kidneys, 444; the heart, 444; meningitic affections, 444; the blood, 444; the skin, 444; globular bacteria, 445; general views, 446; *symptomatology*, 451; the skin, 451; the mucous membranes, 457; organs of digestion, 460; the liver, 461; intestinal canal, 461; the kidneys, 461; cerebral symptoms, 462; the fever, 463; the pulse, 464; complications and sequelæ, 465; pneumonia, 465; pleurisy, 465; peritonitis, 465; menin-

gitis, 466; pyæmia, 466; the joints, 467; gangrene of the skin, 467; thrombosis, 467; the eyes, 468; mental disturbances, 469; influence of the disease upon existing skin diseases, 469; influence on recent wounds, 469; *diagnosis*, 470; differential diagnosis, 470; *stages and duration*, 472; *relapses*, 473; habitual erysipelas, 473; *varieties of the disease*, 474; *mortality and prognosis*, 476; in existing diseases, 476; sex, 476; age, 477; *treatment*, 477; prophylaxis, 477; curative measures, 478; local treatment, 479; expectant treatment, 483; general measures, 483; treatment of complications, 483.
Escherich, 526.
Estlander, 479, 481.
Etiology of dengue, 509; of epidemic cerebro-spinal meningitis, 690; of erysipelas, 425; of hay fever, 542; influenza, 523; of malarial diseases, 563; of measles, 37; of miliary fever, 491; of rubeola, 134; of scarlatina, 161; of small-pox, 326; of varicella, 8.
Eucalyptus globulus in malarial diseases, 669.
Eulenberg, 293, 475, 667.
Eye in epidemic cerebro-spinal meningitis, 707, 718, 726; in erysipelas, 468; in scarlatina, 220, 264; small-pox, 379.
Exanthematic Diseases, Acute, 3; *Introduction*, 3; their common characteristics, 3.

Farr, 160, 324.
Faure-Villars, 711.
Faye, 44, 101.
Feith, 105.
Feltz, 89, 162.
Fenestre, 432, 433, 442.
Fenger, 481.
Fenini, 173.
Fenwick, 211, 227.
Ferber, 491.
Fichtbauer, 99.
Fichter, 670.
Fiedler, 588.
Figueri, 89.
Fitzpatrick, 178.
Fleischmann, 10, 272.
Flemming, 43.
Fleury, 673.
Foderé, 503.
Fontenelle, 584.
Forest, 36.
Forget, 725.
Formey, 181.
Förster, 99.
Fouquier, 47.
Fourcart, 494, 497.
Francis, 307.
Frank, J., 51.
Frank Peter, 597.
Franque, 222.
Frentzel, 696.
Friedreich, 449.
Frölich, 120.
Fuchs, 12, 73, 74.
Fuster, 518.

Gahn, 481.
Galli, 518, 526.
Gallicio, 526.
Galeazzi, Guisman, 559.
Galtier, 490.
Galy, 496, 503.
Gauster, 43, 44.
Geissler, 42, 48, 263, 289.
Gelmo, 133.
Gerhardt, 27, 44, 73, 90, 92, 133.
Gessele, 572.
Gibbs, 618.
Gibson, 436.
Giesler, 640.
Gildemeister, 450.
Gillespie, 185, 189.
Gintrac, 425.
Girard, 172, 626, 627.
Girtanner, 41.
Gläser, 253, 263.
Gley, 104.
Gluge, 516, 524, 526.
Göden, 131.
Good, Mason, 165.
Goodfellow, 435.
Gosselin, 433, 477.
Gonzée, 480, 581.
Grant, 525.
Graves, 44, 233, 432, 522, 529, 597.
Green, 481.
Gregory, 321.
Gresser, 503.
Griesinger, 571, 597, 599, 612, 614, 617, 631, 635, 637, 646, 667, 668, 673.
Grippe, see Influenza.
Griscom, 480.
Grisolle, 673.
Groh, 652.
Groos, 669.
Grooshans, 581.
Gruel, 42, 102.
Gubler, 459.
Guersent, 47, 165, 162.
Gummers, 103.
Gutmann, 196.

Haartman, 43.
Habisreutinger, 47.

de Haën, 43, 630.
Hahn, 120.
Haidenhain, 450.
Haight, 455.
van Halen, 47.
Haller, 431.
Hallier, 163.
Hamburger. 172, 233.
Hambursin, 253.
Hammond, 640.
Hampton, 673.
Hannover, 559.
Hare, 306.
Harley, 228.
Härlin, 173, 216.
Hartmann, 96.
Hasse, 480.
Harvey, 6.
Häser, 529.
Hauff, 174, 233.
Hauner. 90, 92, 99, 100, 121.
Hay Fever, 539; *bibliography*, 539; *etiology*, 542; influence of age, 542; sex, 542; social position, 543; temperament, 543; hereditary tendency, 543; the seasons, 543; emanations from plants, 544; other exciting causes, 544; experiments with the pollen of plants, 545; *symptomatology*, 547; the catarrhal form, 547; the asthmatic form, 549; general symptoms, 550; *diagnosis*, 551; *treatment*, 551.
Headache in small-pox, 346.
Heart in dengue, 513; in epidemic cerebro-spinal meningitis, 706; in erysipelas, 444; in malarial diseases, 627; in measles, 98; in miliary fever, 496; in scarlatina, 233, 255.
Heat, influence of, in malarial diseases, 568.
Heberden, 7, 541.
Hebra, 7, 12, 63, 65. 89, 132, 205, 210, 269, 274, 282, 332, 340, 358, 377, 443, 446, 481.
Hecker, 487, 492.
Hedrich, 51.
Heim, 7, 10, 14, 27, 38, 131, 140, 166, 266
Heller, 707, 722.
Helmholtz. 542.
Hennig, 133, 164.
Henoch, 21, 74, 87.
Hertz on malarial diseases, 555.
Hertwig, 527.
Hervieux, 183, 458, 475.
Herzog, 503.
Heschl, 625, 646.
Heslop, 165.
Hesse, 6, 9, 15, 16, 29.
Heusinger, 580.
Heyfelder, 73, 96, 173, 446, 456, 473.
Higginbotham, 480.
Hildebrand, 105, 131, 165.
Hirsch, 36, 155, 423, 426, 431, 487, 491, 495, 507, 511, 518, 526, 567, 572, 584, 691, 693, 696, 711.
History of dengue, 507; of epidemic cerebro-spinal meningitis, 687; of erysipelas, 423; of influenza, 517; of malarial diseases, 557; of measles, 36; of miliary fever, 487; of rubeola, 130; of scarlatina, 159; of small-pox, 320; of varice
Hochmuth, 69.
Hofmann, 121.
Hoffmann, Fred., 426.
Hoffmeister, 90.
Holm, 462.
Home, 39, 42, 43.
Höring, 52.
Horst, 39.
Horritz, 640.
Huber, 310.
Hufeland, 27, 202, 495.
Huguenin, 226, 228.
Hunter, 429.
Hutchinson, 480.
Huter, 482.
Huxham, 101.
Hydrocephalus in scarlatina, 258.
Hydropericardium in scarlatina, 258.
Hydrothorax in scarlatina, 258.
Hynes, 277.

IMMERMANN, 722.
Incubation of Dengue, 510; of malarial diseases, 587; of measles, 59, 75; of rubeola, 141; of scarlatina, 167; of small-pox, 340; of varicella, 16.
Influenza, 515; *bibliography*. 515; *history*, 518; *etiology*, 522; predisposition, 522; sex and age, 522; atmospheric conditions, 523; exciting causes, 523; contagiousness, 523; march of epidemics, 525; influence of other diseases, 526; among animals, 526; *pathology*, 527; *anatomical changes*, 528; mucous membranes, 528; pneumonia, 528; *symptomatology*, 528; fever, 529; pulse, 529; mucous membranes, 529; cough, 529; digestive organs, 530; nervous symptoms, 530; the urine, 531; the skin. 531; duration of the disease, 532; relapses, 532; variations in the disease, 532; *complications and sequelæ*, 533; *diagnosis*, 535; *mortality and prognosis*, 536; *treatment*, 536; prophylaxis, 538.
Imbert-Gourbeyre, 430.
Iron, tincture of, in erysipelas, 478.

JADIOUX, 214.
Jahn, 131, 192.
James, 481.
Jenner, 401.
Jilek, 570, 657.

Jobert, 481.
Joel, 208, 240.
Joints in dengue, 511 ; in erysipelas, 467
Jones, Handfield, 528.
Jörg, 48.
Jourdanet, 564.

KACZOROWSKI, 482.
Kant, 525.
Kapff, 97.
Kaposi, 29, 474.
Karg, 43, 93.
Kassowitz, 8, 21, 27, 43.
Katona, 39.
Kelber, 381.
de Keghel, 123.
Keller, 669.
Kellner, 103, 111.
Kennedy, 233.
Kentish, 481.
Kesteven, 47.
Key, 629.
Kierski, 107.
Kierulf, 43, 44.
Kidneys in epidemic cerebro-spinal meningitis, 707 ; in erysipelas, 441, 461 ; in malarial diseases, 628, 629, 649 ; in miliary fever, 496 ; in scarlatina, 229, 244, 247, 257 ; in small-pox, 386.
Klebs, 381, 440.
Knapp, 707.
Knecht, 349, 397.
Koch, 44.
Kohn, 29.
Kolb, 298.
van der Kolk, Schröder, 105.
König, 428, 440, 447.
Kopp, 199.
Körber, 16.
Köstlin, 44, 136, 164, 201.
Krauss, 108, 240.
Krieg, 90.
Küchenmeister, 481, 667, 668.
Kuntz, 450.
Kussmaul, 405.
Küster, 133.

LAMBL, 434, 443, 460, 478.
Laborde, 458.
Lacaze, 566.
Lafaye, 90.
Lamotte, 426.
Lancisi, 558, 571.
Landouzy, 531, 455, 496.
Lange, 96.
Langenbeck, Max, 308.
Langer, 452.
Larcher, 434, 444.
Larrey, 481.
Larynx in scarlatina, 222, 279 ; in small-pox, 380.
Lassis, 480.
Laudenbach, 186.
Laudenberger, 212.
Lawrence, 432, 443, 466, 480.
Leavit, 673.
Lebert, 431, 460, 462, 466.
Legrand, 469.
Legroux, 667.
Lentin, 214.
Leo, 349.
Leroy, 162.
Letheby, 166.
Leudet, 534.
Levy, 106.
Lewin, 43, 89, 455, 458, 459.
Lewizky, 667.
Liebermeister, 313, 450, 479, 683.
Lietzau, 466.
Lieutaud, 73.
Lievin, 116, 176.
Lindström, 725.
Lindwurm, 133, 136.
Lippe, 44.
Lisfranc, 480.
Liver in epidemic cerebro-spinal meningitis, 706, in erysipelas, 461 ; in malarial diseases, 612 629, 643, 650 ; in miliary fever, 496 ; in scarl 228, 230 ; in small-pox, 348, 386.
Liverani, 47, 490, 492, 496.
Löbel, 478.
Locatelli, 39.
Lombard, 537.
Look, 40.
Loreau, 494.
Lorenzutti, 672.
Lorinser, 669, 671.
Löschner, 44, 90, 173, 201, 211, 274.
Louis, 476.
Lowenhardt, 15.
Loysel, 485.
Lucae, 707.
Luden, 567.
Lücke, 481.
Luithlen, 43.
Lukomsky, 447. 448.
Lungs in epidemic cerebro-spinal meningitis, 706 in erysipelas, 443, 465 ; in influenza, 528 ; in larial diseases, 611, 627 ; in miliary fever, 49 measles, 93, 95 ; in scarlatina, 223, 279.

MACHIAVELLI, 677.
Maclagan, 164, 425.
Malarial Diseases, 555 : *bibliography.*

history, 557; definition of the disease, 559; *geographical distribution*, 559; *etiology*, 563; soil, 563; heat, 568; the seasons, 569; the rain-fall, 569; the wind, as a vehicle of spreading disease, 571; predisposing causes, 573; race and age, 573; sex, 574; influence of pregnancy, 574; constitution, 575; weakening influences, 575; changes of temperature, 575; previous paroxysms, 576; previous residence, 577; epidemic forms, 577; relation to other diseases, 579; different grades of the disease, 582; nature of the malarial poison, 584; *pathology*, 587; incubation, 587; *general course of the disease*, 588; *simple intermittent*, 588; stage of chill, 590; hot stage, 591; sweating stage, 592; deviations from this course, 593; intensity of the paroxysms, 594; different types, 595; time of attack, 597; intermittent in children, 597; *masked fevers*, 598; neuralgia, 599; other manifestations, 600; *pernicious fevers*, 602; their characteristics, 604; the prodromata, 604; paroxysms, 605; disturbances of the nervous system, 605; cases of apparent death, 606; cerebral disturbance with delirium, 607; eclamptic and tetanic spasms, 608; the hydrophobic form, 608; the choleric form, 608; the pernicious sweating form, 610; the dysenteric type, 610; pneumonic and pleuritic forms, 611; icteric form, 612; gangrene, 613; *remittent and continued malarial fevers*, 614; their caracteristics, 614; the different grades, 616; "la fièvre bilieuse hématurique," 618; malarial cachexia, 620; *pathological anatomy*, 624; blood changes, 624; black pigment matter, 625; the nervous system, 626; lungs, 627; heart, 627; spleen, 627; liver, 627; intestinal mucous membrane, 627; kidneys, 628; the skin, 628; changes in malarial cachexia, 629; the "ague-cakes," 629; *analysis of the individual symptoms*, 630; temperature of the body, 630; changes in the blood, 633; the skin, 636; the circulatory apparatus, 637; the respiratory apparatus, 638; the urine, 638; excretion of urea, 639; other excretions in the urine, 640; dropsy, 641; disturbances of the intestinal canal, 642; the liver, 643; the spleen, 643; the blood the vehicle of the poison, 645; cause of rhythmical recurrence of paroxysms, 646; *course and sequelæ*, 647; *relapses*, 648; kidney diseases as sequelæ, 649; liver diseases, 650; spleen, 650; other derangements, 650; *prognosis*, 650; of the simple fevers, 650; masked fevers, 651; pernicious fevers, 651; the remittent and continued forms, 652; chronic malarial cachexia, 652; *diagnosis*, 652; *treatment*, 655; prophylaxis, 655; prophylactic action of quinine, 657; treatment of the paroxysm, 658; in the pernicious fevers, 660; quinine and its mode of administration, 661, 675, 676; its modus operandi, 667; the eucalyptus globulus, 669; arsenic, 671; its mode of administration, 672; carbolic acid, 672; chloride of sodium, 673; sulphites of soda and magnesia, 673: cold water treatment, 673; alcoholics, 674; chloroform, 674; piperine, 674; other remedies, 674; treatment of sequelæ, 677.

Maillot, 661.

Majer, 175, 186.

Malgaigne, 481.

Malherbe, 443, 461.

Malmsten, 44, 467.

de Man, 133.

Mannkopf, 719.

Manson, 510, 512.

Marchioli, 177.

Marcus, 289.

Marken, 672.

de Marmon, Paluel, 672.

Maroschkin, 673.

Martin, 432, 434, 436, 475, 602, 665, 668.

Martinet, 466.

Martius, 397.

Masarei, 102, 104.

Mascati, 584.

Mason, 100.

Massa, 664.

Mathius, 469.

Mauger, 109.

Maurer, 722.

Mauriac, 469.

Maury, 565,

Mauthner, 43, 121.

Magollini, 673, 675.

May, 184.

Mayr, 37, 40, 46, 47, 62, 63, 65, 101, 104.

McDowell, 480.

Measles, 31; *bibliography*, 31; *history*, 36; *etiology*, 37; nature of contagious principle, 38; vehicles of contagion, 41; susceptibility to contagion, 42; second and third attacks, 42; recurrent form, 45; influence of race, 46; pregnancy in, 46; abortion in, 46; other diseases during, 46; age, 48; sex, 52; epidemics of, 53; seasons of the year, 56; time of infection, 57; length of stage of incubation, 59; *pathology*, 62; anatomical changes, 62; the measle-spot, 62; site of, 64; diversity of form, 64; outbreak of the eruption, 65; duration of maximum of eruption, 67; retrocession of eruption, 67; colorations of skin, result of eruption, 68; desquamation, 70; affection of the mucous membranes of the nose, throat and air-passages, 71; of the conjunctiva, 72; of the tongue, 73; of the mucous membrane of the intestines, 73; of the genital mucous membrane, 74; of the pleura pulmonalis, 74; of the spleen, 74; changes in the blood, 74; *symptomatology*, 75; stage of incubation, 75; prodromal stage, 75; stage of eruption, 78; convalescence, 82; *anomalies of the course*, 83; measles without catarrh, 83; measles without eruption, 84; hemorrhagic measles, 85; variations in the stage of incubation, 87; variations in the prodromal stage, 87; variations in the stage of eruption, 88; *complications*, 90; affections of the skin, 90; the mucous membranes of nose and throat, 91; the larynx, 92; bronchitis, 92; pneumonia, 93; gangrene of lung, 95; diseases of the ear, 95; the mucous membrane of the digestive canal, 96; diseases of the nervous system, 97; the heart, 98; the liver, 98; the genito-urinary apparatus, 99; the glands, 99; acute miliary tuberculosis, 99; diphtheria, 99; ulcerous and gangrenous affections, 100; scurvy, 102; *sequelæ*, 102; influence of constitution, 102; measles predisposing to other diseases, 104; influence of measles upon pre-existing diseases, 104; *diagnosis*, 106; differential diagnosis, 106; *prognosis*, 109; influence of anomalies and complications, 110; age in the prognosis, 111; sex in the prognosis, 113; mortality of measles, 113; *treatment*, 116; isolation, 116; ventilation, 118; general hygienic treatment, 119; treatment of the various symptoms, 119; treatment of fever by cold water, 120; inunctions of oil, 121; quinine, 121; sulphate of magnesia, 122; inunctions of oil of turpentine, 122; treatment of convulsions, 123; of bronchitis, 123; of broncho-pneumonia, 124; of diarrhœa, 125; of epistaxis, 126; of affections of the eye, 126; of the ear, 126; of the larynx, 127; of gangrene, 127; of diphtheria, 128.

Mees, 669.
Meigs, 481.
Meissner, 45, 183.
Menstruation in small-pox, 349.
Mercurialis, 524.
Merkel, 726.
Mertens, 92, 96.
Mettenheimer, 44, 105, 106, 120, 138, 139, 142, 147.
Meyer, 90.
Michael, 631.
Michele, 105.
Michaelsen, 51.

Miliary Fever, 485; *bibliography*, 485; *history and epidemiology*, 487; *etiology*, 491; influence of the seasons, 491; condition of the soil, 492; individual predisposition, 493; exciting causes, 494; communicability, 494; relation of miliary fever to other diseases, 494; *anatomical changes*, 495; decomposition, 496; the lungs, 496; the mucous membranes, 496; the heart, 496; the liver, 496; the kidneys, 496; the nervous system, 496; the blood, 496; contents of the vesicles, 496; *symptomatology*, 497; the eruption, 498;
symptoms, 499; *complications*, 500; *diag*
500; duration of the disease, 501; *relapses*,
mortality and prognosis, 501; *treatment*, 50

Miquel, 162, 183.
Moffat, 572.
Mombert, 105.
Monro, 39, 40.
Moore, 52, 164, 174, 321, 663.
Moreau, 474.
Mortality in epidemic cerebro-spinal meningitis,
in erysipelas, 476; in influenza, 536; in me
113; in miliary fever, 501; in scarlatina,
in small-pox, 391.
Morton, 36, 198.
Mosler, 126, 667, 669, 673.
Moutard, 665.
Mucarin, 673.
Mucous membranes in dengue, 512; in epidemi
cebro-spinal meningitis, 706; in erysipelas,
457; in influenza, 528, 529; in malarial dis
627; in measles, 71; in miliary fever,
rubeola, 141; of digestive organs in scarl
227, 280; of mouth in scarlatina, 219, 27
small-pox, 360, 367, 379, 385; in varicella,
Muhry, 642.
Mühsam, 311.
Müller, 173, 278, 413, 510, 512.
Murchison, 165, 184, 185, 189, 429, 458, 476.
Murray, 495.
Muscular system in scarlatina, 233.

Nasse,
Naumann, 131, 431, 450.
Naunyn, 107
Neisser, 89.
Nervous system in epidemic cerebro-spinal m
gitis, 704, 717, 726; in influenza, 530; in m
diseases, 599, 605, 626, 646, 650; in measles,
miliary fever, 496; in scarlatina, 226, 255
265, 275; in small-pox, 379, 387.
Neuhold, 658.
Neumann, 204.
Neureutter, 95.
Nicola, 44.
Niemeyer, 89, 103, 131, 205.
Nieuwenhuis, 581.
Noirot, 211.
Nolé, 490, 497.
Nunnely, 464, 480.
Nystrom, 481.

Obé, 483.
Obermeier, 349.
Œdema in scarlatina, 257; of the glottis, in er
las, 483; of the glottis, in scarlatina, 258.

Ogle, 164.
Oppolzer, 314, 459
Ornstein, 636.
Orth, 437, 447, 449.
Osiander, 334, 475
Ottoni, 490.
Ozanam, 537.

PAALZOW, 450.
Paasch, 169.
Paget, 185, 667.
Palante, 164.
Pancreas in scarlatina, 227.
Pank, 103, 106.
Panum, 41, 48, 59, 79, 526.
Papoff, 683.
Parent-Duchâtelet, 466.
Parrot, 494, 503.
Pastau, 107.
Pathology of epidemic cerebro-spinal meningitis, 697; of erysipelas, 441; of influenza, 527; of malarial diseases, 587; of measles, 137; of miliary fever. 495; of rubeola, 137; of scarlatina, 202; of small-pox, 340; of varicella, 10.
Patissier, 480.
Paul, 266.
Pepper, 38.
Percival, 39.
Pereird, 665.
Periosteum in scarlatina, 232.
Peter, 443.
Petersen, 164
Petit, 162, 480, 534.
Pezet, 507.
Pfeiffer, 588.
Pfeilsticker, 48.
Pfeufer, 661.
Pfleger, 448, 451.
Phœbus, 541.
Pigné, 503.
Pihan-Dufeillay, 444.
Pinkham, 123.
Pioch, 673.
Piorry, 667, 678.
Pirogoff, 438.
Plonviez, 490, 497.
Polak, 104.
Polli, 673.
Politzer, 88.
von Pommer, 190.
Ponfick, 385, 387, 388, 437, 443.
Poor, 637.
Porter, 277.
Poumier, 618.
Prevot, 274.
Pride, 196.
Priessnitz, 678.
Primbs, 496.
Prognosis of dengue, 513; of epidemic cerebro-spinal meningitis, 723; of erysipelas, 476; of influenza, 536; of malarial diseases, 650; of measles, 109; of miliary fever, 501; of rubeola, 147; of scarlatina, 287; of small-pox, 391; of varicella, 29.
Prophylaxis of erysipelas, 477; of malarial diseases, 655; of small-pox, 400.
Pruner, 109.
Pujos, 434, 439.
Pyæmia in erysipelas, 466.
Pyle, 166.

QUADRAT, 574.
Quincke, 349.
Quinine in epidemic cerebro-spinal meningitis, 737; in erysipelas, 479; in malarial diseases, 657, 661, 675, 676; in measles, 121; in miliary fever, 503; in scarlatina, 306.

RACE, influence of, in small-pox, 329.
Radel, 162.
Ranke, 198.
Ranzoni, 673.
Ratzius, 475.
Rautenberg, 107.
Ravitsch, 447.
Ravn, 100.
Rawling, 537.
Rayer, 43, 73, 105, 424, 426, 467, 494, 496.
Read, 508.
Redtenbacher, 640.
Rehn, 73, 166.
Reil, 289.
Reiter, 413, 414.
Relapses in dengue, 513; in erysipelas, 473; in influenza, 532; in malarial diseases, 648; in miliary fever, 501.
Reuss, 480.
Rhazes, 36, 322, 337.
Rhode, 466.
Rain-fall, influence of, in malarial diseases, 569.
Richardson, 164, 177, 293.
Richeraud, 426.
Richter, 426.
Ricord, 480.
Riecke, 44.
Riess, 163.
Rigler, 109, 609, 627.
Rilliet, 47, 91, 105, 204, 274.
Rindfleisch, 384.
Ringer, 478, 639.
Ritter, 574, 580.
Ritzmann, 431, 434, 435, 439, 443, 465, 467, 469, 473.
Rivière, 6.

Rivolta, 587.
Roberdière, 43.
Robert, 396.
Robinson, 189.
Röthein, see Rubeola.
Röhrig, 450.
Roger, 220.
Rogers, 657.
Rogez, 435.
Roncati, 122.
Rosenstein, 43, 45, 331, 641.
Roser, 106, 175, 438.
Rossi, 39.
Rostan, 162.
Roth, 726.
Rothe, 100.
Roux, 61, 109, 495.

Rubeola (Rötheln, German measles), 129; *bibliography*, 129; *history*, 130; *etiology*, 134; age in, 136; subsequent attacks, 136; contagiousness, 136; epidemics of rubeola, 136; *pathology*, 137; the eruption and its site, 137; the size of the spot, 138; duration of the spots, 139; desquamation, 139; diagnosis between the rubeola and measles spot, 140; *symptomatology*, 141; stage of incubation, 141; the mucous membranes, 141; lymphatic glands, 142; the urine, 143; the stage of eruption, 143; the temperature, 145; *complications*, 147; *prognosis*, 147; *treatment*, 147.

Rufz, 44.
Rühle, 222.
Rush, 507.
Russegger, 250.
Russell, 479.
Rust, 480.
Rüttel, 44.

SABATIER, 469.
Sadler, 101.
Salisbury, 88, 109, 587.
Salvagnoli, 572.
Salzmann, 85, 90.
Sansom, 307.
Sanson, 455.
Santini, 490.
Santlus, 199, 281.
Saussure, 657.
Sauvages, 426.
Savory, 425, 435.

Scarlatina, 149; *bibliography*, 149; *history*, 159; *etiology*, 161; contagiousness of scarlatina, 161; isolation of patients, 161; inoculation of the poison, 162; length of time of exposure to the poison, 164; milk as a vehicle of contagion, 165; spread of the disease by clothing of nurses, 165; animals as a medium of contagion, 166; the
tenacity of the poison, 166; incubation, 16
variations in time of incubation, 168; whe
patient may communicate the disease, 170; ind
vidual predisposition, 172; family predisposit
173; influence of social position, 175; of soil, 17
of locality, 177; age in, 178; sex in, 182; inf
ence of menstruation, 183; of pregnancy, 183;
childbirth, 183; of lactation, 183; of occupatio
183; the disease in those who have undergo
surgical operations, 184; influence of previous di
ease, 185; of race, 185; second attacks of the di
ease, 186; relapse, 190; third attacks, 191; t
recurrent form, 192; sporadic and epidem
forms, 193; the influence of the seasons, 194;
weather, 195; of the drinking-water, 196; e
vation and geological structure of the groun
196; location, 196; condition of the subsoil, 19
personal intercourse in the spread of the disea
197; slaughter-house offal as means of contagi
198; variations in the severity of epidemics, 19
periodicity of epidemics, 199; supposed relati
between scarlatina and measles, 201; pathoge
esis of scarlatina, 202; *pathology*, 202; anato
cal changes, 202; the skin eruption, 202; its s
and general characteristics, 203; description
other authors, 204; progress of the eruption, 2
deviations from the above descriptions, 207; t
miliary eruption, 209; exudations of blood in t
skin, 210; cutaneous diseases following scarlatin
210; post-mortem appearance of the eruptio
211; the desquamation, 212; subsequent desqu
mations, 214; desquamation affecting the nails a
hair, 214; affections of the throat, 214; gangre
of the tonsils, 217; of the connective tissue of t
neck, 217; diphtheria, 217; the tongue, 218; t
gums and mucous membrane of the cheeks, 21
the mucous membrane of the nose, 219; the ey
220; the ear, 221; the larynx, 222; the bronc
222; pneumonia, 223; lymphatic glands, 22
cellulitis of the neck, 225; changes in the brai
226; the mucous membrane of the digestive o
gans, 226; the pancreas, 227; the liver, 228; t
spleen, 228; the genital organs, 229; the kidney
229; the serous membranes, 231; the synovi
membranes of the joints, 232; the periosteu
and bone, 232; the muscular system, 233; t
vascular system, 233; the blood, 234; *differe*
forms of scarlatina, 234; *symptomatology in t*
usual forms of the disease, 237; initiatory sym
toms, 237; the throat, 238; the temperature, 23
the eruption, 239; affections of the lymphat
glands, 242; of the joints, 243; nephritis, 24
the urine, 245; parenchymatous nephritis, 24
irregular course of the disease, 250; angina, 251
cases of scanty eruption, 252; rudimentary sym
toms, 252; cases with blood-poisoning, 253; a

sence of local lesions, 254; *cases of normal beginning and subsequent irregularity*, 255; irregularity of the fever, 255; nervous delirium, 245; intercurrent nervous attacks, 255; sudden collapse, 256; hemorrhagic diathesis, 256; irregularity in the eruption, 256; in the angina, 256; in the inflammation of the cervical glands, 256; in the joint affections, 256; in the results of the renal inflammation, 256; ascites, 257; hydrothorax, 258; hydropericardium, 258; hydrocephalus, 258; frequency of dropsy and albuminuria, 258; date of appearance of dropsy, 259; symptoms of dropsical patients, 259; *cases irregular from the beginning*, 262; high fever, 262; the eruption, 262; the throat, 264; cervical glands, 264; the eye, 264; diarrhœa, 264; kidneys, 264; cerebral symptoms, 265; rheumatic affections, 266; peculiar odor of patients, 266; duration of irregular cases, 267; causes of death, 267; progress in favorable cases, 267; typhoid scarlatina, 268; secondary eruptions, 270; hemorrhagic form of the disease, 272; scarlatina occurring in the course of other diseases, 274; *complications*, 274; nervous symptoms, 275; the eye, 276; the ear, 277; mucous membrane of nose and mouth, 278; cellulitis of the neck, 278; the air-passages and lungs, 279; intestinal mucous membrane, 280; the liver, 280; the spleen, 281; the serous membranes, 281; purulent arthritis, 282; gangrene, 282; *sequelæ*, 283; *diagnosis*, 284; differential diagnosis, 286; *prognosis*, 287; mortality, 291; influence of the seasons, 292; the city and country, 293; poverty and opulence, 293; good nursing and medical advice, 294; influence of sex, 294; influence of age, 294; influence of mortality of scarlatina upon the general mortality, 295; *treatment*, 297; prophylaxis and general sanitary measures, 297; specific and non-specific treatment, 301; bloodletting, 301; cathartics and emetics, 302; anointing with lard, 302; symptomatic treatment, 302; temperature of the room, 303; care of the patient's skin, 303; cold-water bathing, 304; quinine, 306; digitalis, 306; carbolic acid, 307; sulphocarbolate of soda, 307; transfusion of blood, 307; yeast, 307; the warm bath, 307; treatment of brain symptoms, 308; of the angina, 308; diphtheria, 309; the ear, 309; the eye, 309; cellulitis of the neck, 310; isolation of cases of diphtheria and gangrene, 310; bronchial and pulmonary complications, 310; intestinal disturbances, 311; rheumatism, 311; inflammation of serous membranes, 311; hemorrhages, 312; nephritis, 312; dropsy and anasarca, 313; ascites, 314; uræmic convulsions, 314.

Schaper, 335.

Scheby, 349.

Schedel, 426, 469, 581.

Schenk, 36.

Scheich, 537.

Schiefferdecker, 525.

Schneemann, 121.

Schneevoogt, 581.

Scholz, 613.

Schönlein, 12, 131, 264, 425, 426, 462, 503, 525.

Schott, 480.

Schramm, 599, 621.

Schreiber, 100.

Schröter, 220, 276.

Schulze, 103.

Schultze, 44.

Schütz, 44.

Schwalbe, 121, 478, 481, 578.

Schweich, 516, 524.

Scoutetten, 121.

Seasons, influence of, in epidemic cerebro-spinal meningitis, 693; in erysipelas, 431; in hay fever, 543; in malarial diseases, 569; in miliary fever, 491.

Sebastian, 565, 575, 650.

Sedoni, 499, 503.

Seidl, 46, 51, 103, 104.

Seitz, 85, 104, 136, 494, 496, 497.

Semeleder, 459.

Senn, 183.

Sennert, 6.

Sequelæ of epidemic cerebro-spinal meningitis, 721; of erysipelas, 465; of influenza, 533; of malarial diseases, 647; of measles, 102; of scarlatina, 283; of small-pox, 378.

Serous membranes, in erysipelas, 465; in malarial diseases, 611; in scarlatina, 231, 381; in small-pox, 336.

Sex, in epidemic cerebro-spinal meningitis, 696; in erysipelas, 430, 476; in hay fever, 542; in influenza, 522; in malarial diseases, 574; in measles, 52; in miliary fever, 493; in scarlatina, 182, 294; in small-pox, 328, 393.

Sheriff, 509, 512, 514.

Siegen, 669.

Simon, 132.

Simon, G., 63, 205.

Simon, Jules, 459.

Sims, 166.

Skin, in epidemic cerebro-spinal meningitis, 704, 710; in erysipelas, 441, 444, 451; in influenza, 531; in malarial diseases, 628, 636.

Skoda, 668.

Small-Pox, 319; *bibliography*, 319; *history*, 320; *etiology*, 325; influence of age, 326; sex, 328; race, 329; influence of existing diseases, 329; immunity from subsequent attacks, 331; earliest origin and mode of extension, 333; nature of the contagion, 333; the most liable period of infection, 335; infection through clothing, 336; through dead bodies, 336; through gases, 336; conditions favorable to its spread, 338; *pathology*, 340; *symptoms and course of the disease*, 340; incubation, 340; the initial stage, 342; the rigors, 343; temperature, 344; pulse, 344; respiration, 344; appetite, 345; nausea, 345; headache, 346; brain symptoms, 346; pain in the back, 347; the spleen, 348; the liver, 348; the urine, 348; menstruation, 349; the initial eruptions, 349; their frequency, 351; varieties of the initial stage, 352; small-pox without eruption, 352; *hemorrhagic small-pox*, 353; its symptoms, 354; *regular course of the disease*, 355; stage of eruption, 355; stage of suppuration, 357; the mucous membranes, 360; febrile symptoms, 361; stage of desiccation, 363; the itching, 364; the resulting scars of the skin, 364; date of convalescence, 366; *confluent variety*, 366; the mucous membranes, 367; the fever, 368; vomiting, 368; the skin affections, 368; desiccation, 369; *pustulous hemorrhagic small-pox*, 369; *varioloid*, 372; *complications and sequelæ*, 378; the skin, 378; nervous system, 378; the eye, 379; the ear, 379; mucous membrane of nose, 379; the joints, 379; bronchitis, 379; diphtheria, 380; laryngitis, 380; the salivary glands, 380; abdominal viscera, 380; *anatomy of the pock*, 380; changes in the skin, 382; the mucous membranes, 385; the serous membranes, 386; the liver, 386; the kidneys, 386; the spleen, 386; the heart, 387; the nervous system, 387; *diagnosis*, 388; differential diagnosis, 389; *prognosis*, 391; mortality, 391; influence of seasons, 393; of age, 393; of sex, 393; of physical condition, 393; *treatment*, 395; specific methods, 395; treatment of the eruption, 397; diet, 399; complications, 399; *prophylaxis*, 400; *vaccination*, 401; variola of the lower animals, 401; course of vaccinia, 403; its efficacy, 405; length of time of protection, 406; objections to vaccination, 407; transmission of disease, 409; performance of vaccination, 411; preservation of vaccine virus, 412; choice of patients, 413; the operation, 414; compulsory vaccination, 416.

Smart, 596.

Soda, sulphocarbolate of, in scarlatina, 307.

Soil, influence of, in malarial diseases, 563; in miliary fever, 492.

Solon, Martin, 480, 492.

Spleen in epidemic cerebro-spinal meningitis, 706, 710; in erysipelas, 441; in malarial diseases, 627, 629, 643, 650; in measles, 74; in scarlatina, 228, 281; in small-pox, 348, 386.

Spengler, 481, 525.

Speranza, 39.

Spiess, 42, 43, 44, 52, 99.

Squire, 133.

Stäger, 280.

Starcke, 477, 596.

Steffen, 318.

Steiner, 73, 95, 244, 258, 475.

Steinthal, 99.

Steudel, 99.

Steudener, 444.

Stevenson, 186.

Stewardson, 628.

Stiebel, 43, 44, 74, 199, 253, 266.

Stiener, 396.

Stilling, 44.

Stofella, 78.

Stoech, 596.

Stratton, 185.

Struve, 670.

Suppuration, stage of, in small-pox, 357.

Sydenham, 36, 198, 337, 346, 478, 558.

Symptomatology of dengue, 510; of epidemic cer spinal meningitis, 707; of erysipelas, 451; of arial diseases, 558; of miliary fever, 497; of fever, 547; of influenza, 598; of measles, 7 rubeola, 141; of scarlatina, 237; of small-pox, of varicella, 15.

Synovial membranes of joints in scarlatina, 232.

Swiney, 281.

Taglioni, 665.

Talbor, Robert, 558.

Tarchanoff, 668.

Taube, 230.

Taupin, 105.

Taylor, 165.

Teilhol, 492.

Tessier, 566, 672.

Testa, 103.

Thaer, 120.

Thaly, 509, 511.

Thaulow, 44.

Theden, 496.

Themmen, 39.

Thierfelder, 138, 142, 147.

Thoinnet, 443.

Thomas, 464, 673.

Thomas on acute exanthematic diseases, 3; on me 31; on rubeola, 129; on scarlatina, 149; on cella, 5.

Thompson, 274.

Thomson, 7.

Thore, 90, 96.

Thoresen, 41, 166.

Throat in scarlatina, 214, 298.

Thrombosis in erysipelas, 467.

Thuessink, 39, 41.

Tigri, 529.

Titeca, 696.

Tomasi, 629.
Tongue in measles, 73; in scarlatina, 218.
Tonsils, gangrene of, in scarlatina, 217.
Torti, 558.
Tourner, 665.
Tourtual, 183.
Townsend, 261.
Treatment of dengue, 514; of epidemic cerebro-spinal meningitis, 733; of erysipelas, 477; of hay fever, 551; of influenza, 536; of malarial Diseases, 655; of measles, 116; of miliary fever, 502; of rubeola, 147; of scarlatina, 297; of small-pox, 395; of varicella, 30.
Tresling, 43.
Treulich, 672.
Triboulet, 101.
Tripe, 133.
Trousseau, 13, 16, 21, 27, 125, 126, 133, 167, 183, 253, 311, 358, 425, 427, 427, 431, 438, 443, 458, 459, 460, 474, 667.
Trojanowsky, 43, 45, 187, 189.
von Tscharner, 165.
Tuffnell, 61.
Tüngel, 78.
Turner, 673.
Tutschek, 467.

UHLE, 640.
Upham, 711.
Urine in dengue, 512; in erysipelas, 460; in influenza, 531; in malarial diseases, 638; in measles, 31; in miliary fever, 499; in scarlatina, 245; in small-pox, 348.

VACCINATION, 401.
Valleix, 398.
Varicella, 5; *bibliography*, 5; *history*, 6; *etiology*, 8; *pathology*, 10; anatomical changes, 10; the vesicles, 11; their appearance, 11; duration, 13; *symptomatology*, 15; incubation, 16; eruption, 16; temperature, 19; relapses, 20; affection of mucous membranes, 21; *diagnosis*, 22; *prognosis*, 29; *complications*, 29; *treatment*, 30.
Varioloid, 372.
Vauquelin, 584.
Vanory, 508.
Veale, 133.
Veillard, 619, 628.
Veit, 42, 69.
Velpeau, 443, 459, 466, 480, 481.
Veratrum in erysipelas, 478.
Verneuil, 495, 499.
Vetter, 10, 27.
Vézien, 44.
Viennois, 410.
Vieusseux, 711.
Virchow, 444, 446, 450, 457, 475.
Vogel, 38, 140.
Vogel, A., 133, 474.
Vogel, S. G., 51, 186.
Voit, 99.
Volkmann, 425, 427, 436, 438, 439, 444, 458, 462, 466, 469, 471, 473, 479, 480.
Volz, 73, 172.

WACHSEL, 39.
Wagner, 133, 385, 386, 388, 469.
Wagner, E., 228.
Walz, 104, 121.
Water, cold, in erysipelas, 479, 481; in measles, 120; in scarlatina, 305; in small-pox, 398.
Waton, 608.
Ward, 600.
Weber, 73, 229, 282.
Weber, H., 283.
Webster, 43.
Weigert, 381.
Weil, 77, 96.
Weisse, 46, 104, 105.
Wells, 432, 580.
Wendt, 40, 44, 103, 126, 221, 277, 309.
Wenzel, 566, 569.
Werlhof, 559.
Wernher, 480.
West, 127, 132.
Westerland, 481.
Willan, 7, 39, 132, 166, 186, 436.
Wildberg, 263.
De Wilde, 507, 511, 514.
Williams, 166.
Wilkes, Samuel, 465.
Wilkinson, 478.
Wilks, 444.
Wilson, 44, 73.
Wise, 507.
Wisshaupt, 78.
Wittwer, 524.
Wood, 177, 282.
Woodbury, 527.
Wunderlich, 44, 133, 265, 280, 429, 463, 665.
Wutzer, 443, 459, 481.
Wyss, 385.

ZEHNDER, 175, 260, 263, 278, 282.
Zengerle, 163, 166.
Ziemssen, 121, 124, 313; on epidemic cerebro-spinal meningitis, 683.
Zimmermann, 631, 639, 659.
Zuccarini, 430, 443.
Zuelzer on dengue, 505; on erysipelas, 419; on hay fever, 539; on influenza, 515; on miliary fever, 485.
Zülger, 164, 324, 337, 341, 381, 388, 397.
Zürn, 166.